

Alice in Wonderland Syndrome

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Jan Dirk Blom

Parnassia Psychiatric Institute, The Hague, The Netherlands

Leiden University, Leiden, The Netherlands

University of Groningen, Groningen, The Netherlands

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1. Introduction

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

No-one wants to be ill—and yet, some disorders have such intriguing names that they inspire more than just aversion. For me, ‘Alice in Wonderland syndrome’ is such a name. It conjures up images of golden afternoons in a beautiful garden filled with bright flower beds and cool fountains where the Cheshire Cat appears and disappears with a grin and Hatter and Hare drink tea while the Dormouse sleeps. It certainly sounds better than ‘cardiac arrhythmia’ or ‘pneumonia’, to mention just two other afflictions. Even so, names can be deceiving. Therefore, let’s consult someone with first-hand experience before we accept too readily that Alice in Wonderland syndrome is as fun as the name might suggest.

Let us ask Ms. Artemis¹, for example: a bright, intelligent woman of 20-something who came to see me on a balmy summer day at my outpatient clinic in The Hague. Some months beforehand, she had been using a combination of amoxicillin, clarithromycin (both antibiotics) and pantoprazole (a proton pump inhibitor used to decrease the amount of acid produced in the stomach) to treat a *Helicobacter pylori* infection that had been causing her nausea and discomfort for over 2 years. As the nausea had subsided within a few days, she was glad that her family physician had given her the prescription, and, for the first time in many months, she was able to go to work without any physical ailments. However, 3 or 4 days into treatment, she had woken up to find that she was seeing everything through

a purplish haze that gradually faded over to orange at the top of her field of vision.

Chromatopsias such as these are rare but well-known side effects of antibiotics. I had never before heard of that particular combination of purple and orange, whereas during treatment with such medicines, seeing things in a single hue for a while does happen occasionally. However, what Ms. Artemis experienced next was even stranger. Whereas Alice had had the White Rabbit to guide her down into Wonderland, what Ms. Artemis got was a squirrel: an orange-brown, life-size squirrel that was sitting opposite of her in an empty seat on the morning train, quietly gazing into the distance. She stared at it with a mixture of surprise and bemusement, wondering how such a shy little animal might have wound up in this train coach. Squirrels are native to the Netherlands, but their numbers are small, and, in rural areas, they are rarely sighted, let alone in train stations or on coaches.

The squirrel stayed put—but when another traveller passed between the two of them, it disappeared. Ms. Artemis got off the train wondering where it had gone, but it did not take long for it to reappear. In fact, it did so several times that same day, in a garden, on her desk at work and in many other places where squirrels are not usually found. Whenever she saw it, it was right in front of her and stayed there each time for a duration of about 30–60 s (or so she guessed afterwards). Ms. Artemis was well aware that no-one else reacted to the squirrel, and, therefore, no-one else appeared to be seeing it. She tried to touch it several times—however, whenever she extended her arm, it invariably disappeared. Having read the information leaflet accompanying her medication, she decided that this must be some sort of hallucinatory side effect of the antibiotics she was using. This was something I was able to confirm by the time she had come to consult me (although I suspected that the proton pump inhibitor, rather than the antibiotics, was to blame). Ms. Artemis stopped taking the medication, and, one week after they had entered her life, both the squirrel and the colours disappeared without any further intervention.

Nevertheless now, in their place, a new phenomenon presented itself: again, for a duration of 30–60 s, she started to experience ‘spells’ (as she called them), during which she saw the objects on her desk rise silently into the air, all the way up to the ceiling, where they would start to rotate around each other. These episodes ended as abruptly as they had begun, and,

afterwards, Ms. Artemis' mental acuity was always clear. All that she felt in the aftermath was a slight headache near the left temple, not anything even coming close to the pain of a classic migraine .

If the squirrel had been a remarkable phenomenon, by comparison, the floating and gyrating objects were something truly peculiar, something that her family physician and neurologist had never encountered before. And neither had I—not once during two decades of intensive contact with psychotic patients. Nevertheless, it was clear to me that this was neither illusion nor hallucination.

Hallucinations are percepts, experienced during wakefulness, which lack an appropriate source in the external environment. Seeing a squirrel that isn't there (as Ms. Artemis had done), feeling a hand upon one's shoulder while there is no-one around and hearing a voice when one is all alone are common examples of hallucinations. Illusions , on the other hand, do have a source in the external world—but one that is either misperceived or misinterpreted. Perhaps, like me, while walking in the dark, you may have caught a glimpse of someone stepping out of the bushes, only to find that it was a branch or a plastic bag moving in the wind. Similarly, you may sometimes have heard music in the drone of an air conditioner or in the sound of your computer fan—or, when you are a parent, the voice of your child calling out your name. I have experienced all these types of illusion. None of them ever lasted very long. In all cases, there was something out there that evoked them, and, in all cases, it took maybe a second or so for me to realise what had caused it. Occasionally, illusions may last longer—for example, when we discern a face or an animal in a cloud or a coffee stain; but, even then, we are well aware what evokes the image, and we never confuse it for what it really is.

What Ms. Artemis had experienced, however, was something different. During her 'spells', she had been seeing things that were really there but in a way that no-one ever experiences them under normal circumstances. Their position in space seemed to alter, and their position relative to each other seemed to alter, turning around each other at the top of her field of vision. Moreover, there was no way for her to figure out what was really going on. No illusion, no hallucination—but something entirely different.

What Ms. Artemis had experienced is what we call a metamorphopsia . This term comes from the Greek words *metamorphoun* (to change the form) and *opsis* (seeing). It translates as 'visual distortion'. This type of

misperception is conceptually different from illusion and hallucination and is considered one of the hallmark signs of Alice in Wonderland syndrome. In the literature, over 40 different types of metamorphopsia have been described. Thus, people may see things as smaller than they are (micropsia) or as larger (macropsia); they may perceive stationary objects as if continually receding into the distance (porropsia); they may perceive everything as slanted (plagiopsia); they may be unable to properly perceive any movement (akinetopsia); they may see multiple images trailing behind a moving object (trailing phenomenon); and so on. Ms. Artemis' type of metamorphopsia is called gyropsia (i.e. 'seeing circular movement'). Thus, what she experienced during those spells of hers was gyropsia, which is a type of metamorphopsia which, in turn, is considered a symptom of Alice in Wonderland syndrome.

Was it fun for Ms. Artemis to have this? As you may have guessed, it certainly was not. When symptoms of Alice in Wonderland syndrome are mild and transient, people may not be bothered too much by them. They may even be intrigued—as people sometimes tell me. However, when they experience them on a scale such as Ms. Artemis did, they tend to lose confidence in the world around them and start to fear that everything may suddenly collapse. They may also fear that they have dementia or schizophrenia and may end up in a psychiatric hospital, or worse. In that sense, Alice in Wonderland syndrome may be really burdening. Similar to what happened to Alice in the story by Lewis Carroll , it can make us feel much larger than we are and, at other times, much smaller. It can make us experience our legs as shutting up like a telescope or even make us see our hands change into paws and our faces into grotesquely distorted masks when we look in the mirror. Stationary objects may appear to be moving, buildings on the left may be mislocated as standing on the right, and colours may be either hypersaturated or bleed away to turn everything into a dull grey. The name may sound like fun, but Alice in Wonderland syndrome is not about golden afternoons and tea with friends who speak in amusingly convoluted riddles. As the old adage goes, we'd better be careful what we wish for. Moreover, we may search the planet for a renowned specialist—but good luck with that. In many places, neurologists will refer you to a psychiatrist, and most psychiatrists will, in turn, ask you what you think about it yourself, and then you'll probably end up pleading for a referral back to the neurologist.

Over the past 10 years, the number of scientific publications on Alice in Wonderland syndrome has doubled. Although that may sound as if the topic is now in the centre of international scientific attention, that is not the case. When I began writing this book, the number of peer-reviewed scientific papers on the subject was 70: only 70 papers written over a time span of 60 years. That boils down to 1.1 paper per year (rounded upwards, mind). Moreover, most of those papers were case reports and modest case series, not anything aimed at creating a synthesis or an overview. Therefore, in 2016, I wrote a review paper [1] and a book chapter [2] to fill that gap. It was while working on those texts that I realised that there was so much more to be said about Alice in Wonderland syndrome that a book seemed mandatory. As you can see, the result is not an impressively thick book. Nevertheless, it is still thickish for what many scholars consider a mere footnote in the neuroscientific literature. After all, Alice in Wonderland syndrome was, until recently, believed to be so rare that most university courses in medicine, psychology and the neurosciences did not even bother to address it.

If you simply wish to gain a quick-and-dirty impression of what Alice in Wonderland syndrome is, the Internet boasts an impressive number of easily accessible synopses. The beauty of the Internet is that it brings an unprecedented load of information within the reach of anyone who has access to a computer, tablet or smartphone. The downside, however, is that the sources are not always explicitly stated and that the quality of what we find is ... well, how should I put this diplomatically? Let me just say that it is wise not to believe everything we find out there. It is not my intention to downplay the usefulness of popular science sites, but Wikipedia², for example, informs us that Alice in Wonderland syndrome is ‘a disorienting neurological condition that affects human perception’ (correct); that it is ‘also known as Todd’s syndrome, or lilliputian hallucinations ’ (both wrong); and that its ‘hallmark sign’ is ‘a migraine ’ (a case of bell and clapper).

Some people who suffer from migraine do indeed experience symptoms of Alice in Wonderland syndrome, either before, during or after an attack—so at least there is a kernel of truth in the latter statement. However, there are numerous other conditions that cause these symptoms, and under no circumstances does migraine constitute ‘a hallmark sign’ or even ‘a sign’ of Alice in Wonderland syndrome. That would be equivalent to saying that ‘a

broken leg' is a hallmark sign of 'pain'. It is impossible, it is meaningless and, frankly, so profoundly nonsensical, that it might well have been a line out of *Alice's Adventures in Wonderland*.

Obviously, Carroll's book is in no need of an introduction. In the Western world, at least, it has become ingrained in the very fabric of our culture (Fig. 1.1). I remember reading a Dutch version of it as a child (one of 174 languages into which the book has been translated) and, like so many people before and after me, was deeply impressed by it. Later, while I was studying medicine in Groningen, I read the English version from Chancellor Press [3], and, after that, I reread it many times, discovering with each reading new insights and delights. However, most rewarding was reading the book with my daughter, Esther, when she was 10 (the age of the real-life Alice Liddell when Carroll wrote the book for her). The story wasn't new to Esther, as she had watched the Disney animated version as well as the live-action spin-off by director Tim Burton , with Mia Wasikowska as Alice and Johnny Depp as the Mad Hatter [4]. Plus, we both like to hum along with *Alice's Theme* by Danny Elfman [5], so Alice was never far away for us.



Fig. 1.1 *Alice in Wonderland*, oil on canvas by George Dunlop Leslie (1879). The girl's dress invokes associations with Alice, but at the time even the book with the characteristic red-cloth binding made the painting's theme instantly recognisable

Nevertheless, Esther was not satiated by all things Alice that she had consumed. I was surprised to see how much she was into the written version and how she responded to Alice, the characters she meets, the twists of the story, the dream logic, the surreal puns and everything else that goes on in the book. She had always loved being read to, but *Alice* was genuinely hard to put down—for the two of us. As Esther told me, the characters were not at all like she remembered them from the films. Apparently, her brain was capable of summoning images that were untainted by the ones she had already seen—which says something about her powers of imagination, but no less about the book's power to speak for itself. And that after a century and a half, across a time gap during which the world has changed so much that children grow up with iPads and laptops rather than with books made of paper with funny drawings in black and white. That definitely says something about the book.

On the other hand, what may need a proper introduction is Alice in Wonderland syndrome, the subject of the present book. Even though there may be a familiar ring to the name and many people have at least heard of it, its characteristics and causes have remained elusive to the greater public. I cannot blame Wikipedia for depicting the syndrome the way it does, as it is barely known to many neurologists and psychiatrists, i.e. the specialists most likely to encounter it in clinical practice. After I had submitted the manuscript for my review paper to the scientific journal *Neurology*, one of the reviewers wrote, ‘I cannot recall a single case of ... AIWS in my 26 years of practice’. It wasn’t hard to imagine the reviewer, a seasoned and obviously highly respected neurologist (otherwise, he or she would not have been invited to review), sitting behind a computer in some far-off hospital with an expression of mild despair, thinking, ‘Either I have managed to systematically overlook this condition in all my years of practice, or the author is a total fantasist’. Since the paper did get published then, apparently, the American Academy of Neurology (who issues the journal) did not consider its contents to be a flight of fancy. Nevertheless, the stories of individuals diagnosed with this syndrome would hardly be out of place in a fantasy novel or a fairy tale. Take Ms. Artemis’ story, for example, and tell me without blinking that it would not have been fit for a modern-day sequel to the original *Alice* story.

Incidentally, not all symptoms of Alice in Wonderland syndrome are visual in nature. As we saw, they can also present as distortions of the way we experience our body—a group of phenomena known as somesthetic distortions. We all remember how Alice grew alternately shorter and taller during her stay in Wonderland; such apparent changes of body size can be experienced for real; we call them microsomatognosia and macrosomatognosia, respectively [6]. Like metamorphopsias, these bodily symptoms are traditionally considered to be very rare—but I think they might not be so rare after all, as I know several people who had similar experiences. Moreover, I myself once had them while I was down with a fever in Thailand.

In 1992, I had been travelling with my then-future wife, Renate, with the Trans-Siberian Express from Moscow to Beijing and from there with all kinds of local transportation throughout China. We had marvelled at the architectural wonders of the Forbidden City, lost ourselves in Beijing’s hutongs, eaten noodle soup and meat of indeterminate animal species under

roofs of corrugated iron, slept with ear plugs in noisy hotels, climbed Huangshan Mountain amidst hordes of Overseas Chinese who wished to see a halo around their heads when the sun came up³, fled from a nest of snakes in a bamboo forest, slept on a bench in a train station near the river where the railroad tracks had unexpectedly come to an end and ridden a bicycle in Dali ('feel the wind in your hair, feel the sun in your face, rent a bicycle', read the English-language sign on the wall of *Charlie's Bicycles*)—and because little else was in English and all the rest was quite unintelligible to us, we had experienced such a profound culture shock that when we reached the southernmost part of China, we felt too overwhelmed to take in the sight of yet another temple or have yet another incomprehensible argument with some well-meaning transportation official. All we wished for was a quiet place that would allow us to digest the experiences from our trip before we would be turning back to the professional demands that were lying in wait for us in Holland.

That place we found in Thailand, on a remote beach on the island of Ko Pha Ngan, which at the time could only be reached from the harbour by boat or (via a network of convoluted dirt tracks through the island's thick jungle) by motor cycle. We opted for the boat ride and, to our great delight, found a beach so pristine that we couldn't believe our eyes. White sands, crystalline water, coconut palms as far as the eye could see, a dozen idyllic bamboo huts in their shadow and only the locals and a handful of backpackers to share them with. It was there that we recuperated from the assaults that beautiful, yet noisy, dirty and inscrutable China had made on all our senses. We literally dusted ourselves off, got rid of our coarse travelling clothes and for a whole week wore hardly anything but a sarong over our bathing suits—in which we practically lived, as we got in and out of the water all day. I shaved off the beard that I had grown on the road, floated silently in the emerald waters of the bay and basically did nothing but eat, sleep and hang out with Renate, indulging in the peace and calm of this heavenly place.

So far, so good—until we both ate something bad and ended up in our bamboo hut with a high fever and a lot of puking over the balcony onto the incredibly soft white sand underneath.

It was there, lying on my back, glowing and shivering in the half dark, that I felt my hands growing to the size of boxing gloves. The sensation was oddly familiar, as if I had experienced it before as a child—and yet it took

me completely by surprise. When I looked at my hands, there was nothing out of the ordinary to be seen, not even the slightest hint of a swelling. But as soon as I laid them down and broke the visual confirmation of their actual size, I felt them regaining their previous outrageous proportions.⁴

The technical term for that sensation is *partial macrosomatognosia*. In Wonderland, Alice experiences something similar when her neck grows incredibly long (so long indeed, as Carroll tells us, that it sticks out above the trees), although in other passages she undergoes *total-body* macrosomatognosia and *total-body* microsomatognosia, meaning that her body as a whole becomes taller and shorter, respectively. In my case, it was only the hands and, fortunately, the sensation lasted only as long as the fever. That is often the case with Alice in Wonderland syndrome, that symptoms are present only briefly, and vanish as soon as the underlying cause is taken away. Sometimes, however, they may last longer. One of my patients in The Hague, Mr. Salvatore, has experienced a sensation that is the opposite of the one I had: whenever he puts his hands in his pockets, he feels them shrinking to the size of tiny stumps—and he has had that recurring sensation for over 35 years.

So, the symptoms of Alice in Wonderland syndrome may be either visual or somesthetic in nature, affecting the way we see the world and/or the way we experience our body. However, there is more ... You may never have considered your sense of time as a sensory modality, but obviously, we all *perceive* time while it appears to be going slower or faster, depending on the extent of our engagement with any given situation. When we are bored, time tends to drag; when we are thrilled, it seems to fly. That sense of time passing is what we call psychological time. It is the counterpart of chronological time, which is measured by clocks and other chronographs, always at the same pace, unaffected by whatever circumstances (that is to say, on Earth at least, although even here, time is affected by altitude, with clocks at sea level going a fraction slower than those high up in the mountains) [8]. Psychological time, on the other hand, is subject to considerable change. When it changes dramatically, we speak of a 'time distortion'. Such distortions can be profoundly disorienting. One of my patients once told me that when he walked from the hospital towards the bus stop on the corner of the street, one moment everything would be normal, the next all things would slow down so as to almost freeze, and then everything would speed up, and cars and bicycles would zip past him,

giving him the feeling that he himself were moving in slow motion—and back again, alternating between incredibly slow and incredibly fast.

Another patient of mine, whom we had admitted to our psychiatric hospital, complained that he had lost all sense of time passing. Whenever he sat in his room in the nursing ward, he had to look at his phone to see how much time had passed, having no idea whether he had just finished breakfast, whether it would be time for lunch, or whether the nursing staff had perhaps forgotten to check in on him for the remainder of the day.

Another symptom of Alice in Wonderland syndrome is derealisation . It shouldn't take much effort on your side to imagine that alterations in your sense perception, such as those described above, may have a deeply alienating effect on you. At least they had on me, when I was lying in that bamboo hut with my hands the size of boxing gloves. Try to imagine, by way of a mental experiment, what the world would look like if you were unable to see any vertical lines. Look around you and try to imagine your surroundings without such lines. What would it look like? Is it even possible to imagine a world like that? And how would you describe it to others? Alternatively, try to imagine what the world would look like if you were unable to see any ridges or wrinkles, as in *arugopsia* , another type of metamorphopsia . With increasing age, the prospect of seeing oneself in the mirror without any wrinkles may seem enticing, but the truth is that arugopsia makes everything look plasticky and artificial and that people experiencing it tend to feel profoundly alienated.

Experiencing the world as unreal, a phenomenon known as derealisation, can be the net result of such perceptual distortions . Things don't look the way they did, and, as a consequence, they affect us differently from the way they did before. Our surroundings appear to be changed, subtly yet profoundly. This may lead to estrangement from the world and also from those we normally share it with, because one of the major problems with Alice in Wonderland syndrome is that people experiencing it have a hard time explaining it to others. (With that, I don't mean explaining it in terms of brain processes but in terms of what the experience is like, i.e. its phenomenological quality.)

To Mr. Salvatore, whose hands repeatedly felt like tiny stumps, derealisation constituted his chief complaint. This was worse for him than feeling his hands decrease in size—especially since, in his case, the feeling of derealisation was accompanied by *depersonalisation* , meaning that he

also lost his sense of self. As he told me, sometimes that experience was so overwhelming that when he rode his bicycle through town, he had the feeling that no-one was actually riding it. Feel the wind in your hair? The sun in your face? Not Mr. Salvatore, during such moments.

In the *Alice* story, perhaps the Cheshire Cat comes closest as an analogy to this distressing experience. If you ever saw him, you will certainly remember him—the Cheshire Cat, as depicted in the original book: sitting on a tree branch, smiling his famous Cheshire-Cat smile and slowly disappearing from back to front (Fig. 1.2), so that on the next page only his smile is left for us to see (Fig. 1.3). A smile without a cat. A bike without a cyclist—the only difference being that the Cheshire Cat just keeps on smiling, whereas for Mr. Salvatore, there was nothing to smile about when he felt the way he did.



Fig. 1.2 *Alice looking at the Cheshire Cat*, illustration by Sir John Tenniel (1890)



Fig. 1.3 *Disappearing Cheshire Cat*, illustration by Sir John Tenniel (1890)

Story elements such as the Cheshire Cat and Alice's bodily changes make one wonder whether Lewis Carroll had perhaps experienced phenomena such as these himself. At least it made me wonder. How else could he have known about them, I thought, and for what other reason would he have bothered to include them in a story intended for children? We now know that many passages in the *Alice* story are thinly disguised events from Carroll's real life in Oxford and that some of the characters in it are based on people who inhabited the academic bubble in which he and the real Alice (Alice Liddell) used to live. Could it therefore be true that the curious perceptual phenomena described in the *Alice* story had a similar source in the author's life? Seeking to answer that question will be another thread in this book.

Few authors have been subjected to as much scrutiny as Lewis Carroll—whose real name was Charles Lutwidge Dodgson (Fig. 1.4)—and on few authors has been written so much by so many. Throughout the world, there are Lewis Carroll societies , with members collecting and exchanging

information, organising meetings and issuing journals with appropriately playful names such as *The Looking-Glass Letter*, *The Carrollian* (formerly called *Jabberwocky*), *Knight Letter*, *Bandersnatch*, *Mischmasch* and *The Lewis Carroll Review of Books*, while papers on Lewis Carroll are also occasionally still published in peer-reviewed scientific journals. And then there are books, dozens and dozens of books, on Charles Dodgson, his life in Oxford, his relation with Alice Liddell and her family, his prose, his poetry, his photographs, his mathematical works, the significance of numbers in his works, hidden layers of esoteric wisdom in the *Alice* story, Alice Liddell herself, semiotics and linguistics in Alice's world, fantasy stamps and envelopes sent through the Wonderland post; you name it, and there is a book written about it. There is even a book on Dodgson's books: not the ones that he wrote, but the ones that he had collected and read [9]. And, lest I forget, a three-volume collection of translations of *Alice's Adventures in Wonderland* into 174 languages, complete with essays on the languages at hand, and foreign translations of 'A Mad Tea Party' translated back into English to see what was made of Carroll's word plays, puns and twists of meaning [10].



Fig. 1.4 Charles Lutwidge Dodgson at age 50, holding a lens of his photo camera; photographed by Oscar Gustave Rejlander (1883)

During my quest for clues about Dodgson's medical history, I soon found myself sucked into this world of Carroll studies and absorbed by what each scholar had to say about this enigmatic figure. Before long, my mind began to conjure up the contours of the person that Charles Dodgson must have been and reading about him shed light on numerous aspects of his life and personality. Nevertheless, he appeared to have a certain sphinx-like quality that made it hard to get him sharply into focus. As a result, reading Carroll biographies soon became a compulsion. Since I found that each biographer told the story from a slightly different angle, highlighting aspects that others had suppressed, attaching meaning to things that others had considered trivial and producing facts that no-one else had done before,

the image of the man as it had begun to form itself in my mind was constantly challenged by what others had to say, thus forcing me to adjust that image with each reading. The only comparable experience I had had in this respect was with works of fiction wherein a single event is told from the vantage point of different narrators, as in Milan Kundera's *The Unbearable Lightness of Being* [11], where each subsequent rendition of Tomas' short life leaves one with a version that is richer and more rewarding than the previous one, and David Lynch's *Twin Peaks* [12], in which each episode shines a different light on the people who had been present during the night that Laura Palmer had been murdered. Likewise, reading Carroll biographies gradually became like revisiting my favourite thriller over and over again, each of them feeding me new clues to solve the mysteries at its heart.

In short, by venturing to go into the story behind the *Alice* book, I had stumbled upon one of the richest collections of biographical material on a single author, put together by a veritable legion of scholars. However, despite all this biographical, historical, philosophical, critical, esoteric and literary material—not to mention the integral publication of what is left of Dodgson's diaries and letters—it soon dawned upon me that the question of whether the man himself might perhaps have suffered from the syndrome that was named after his famous protagonist had never been answered satisfactorily.

Not that others had not tried. During my research I came across several interesting hypotheses, some of which were furnished with seemingly incontrovertible evidence of how things must have gone down with Charles Dodgson and his alleged perceptual distortions. All these hypotheses had been proposed by eminent authors, many of whom were, moreover, well acquainted with the cultural and historical niche that Dodgson had inhabited, thus giving them a head start over someone like me, a non-Brit and non-Carrollian, who had merely visited the UK a handful of times and Oxford only once. What could I possibly add, I thought, to what these learned men and women had already said? What could I, the outsider, say about this thoroughly British affair?

Perhaps not too much.

Perhaps nothing at all.

Once I had realised that, I thought I might as well turn my disadvantage into an advantage by offering exactly that, the perspective of the outsider:

the outsider who lacks a natural acquaintance with the British way of life, now or during the Victorian era; the outsider who lacks the sensitivities that come with being an Englishman, an Oxfordian, a Carrollian; the outsider, in short, who is not hindered by any preconceived notions about the subject matter at hand.

What I seek to do in this book, therefore, apart from outlining Alice in Wonderland syndrome and explaining what is known about its neurobiological underpinnings, is to offer a re-examination of Dodgson's life and work from the perspective of a continental psychiatrist, rather than that of a British scholar or a Carrollian. As a psychiatrist, I am primarily interested in positive disorders of perception. That group of disorders comprises hallucinations, illusions and distortions. As a consequence, much of my scientific work is on the neurology of perception. It is from that vantage point that I intend to approach *Alice's Adventures in Wonderland*: not to make it the subject of psychoanalytic interpretation, as one might perhaps expect from a psychiatrist, but to find out which clues lie hidden in Carroll's work that hint in the direction of an Alice in Wonderland syndrome *avant la lettre*.

For what follows, I am grateful to John Todd, the psychiatrist who first described the Alice in Wonderland syndrome, without whom we might still be very much in the dark about what it means to experience perceptual distortions. Secondly, I am indebted to all those scholars who collected information about Charles Dodgson's life and work. Without them, the task of reconstructing the man's medical history would have been like doing all the running I could do, without ever getting anywhere. Furthermore, the present book—and in fact most of my work—would have been impossible without the many patients with Alice in Wonderland syndrome whom I encountered over the years. We will meet several of them on paper and hear what they have to say about metamorphopsias, somesthetic distortions, derealisation and all those other mind-boggling experiences that are part of the syndrome. It is to them that I owe my deepest gratitude. From those with whom I stayed in touch, I obtained written permission to include their remarkable stories. As to those whom I was unable to trace after they had consulted me (some of them many years ago), I hope they will agree that their stories are worth recounting for the purpose of advancing our knowledge of this neglected condition and bringing it to the attention of the greater public. I have taken care to render their accounts as faithfully as

possible while seeking to minimise the chances that any specific details might reveal their true identities.

There are various questions that will be addressed in this book. First and foremost: What is Alice in Wonderland syndrome? How do we recognise it? What causes it? Is it substance-induced, as some people say it is? Is it heritable? How can it be treated? And, most importantly perhaps, does it always need to be treated? In addition, I will address the broader questions of what the syndrome tells us about regular sense perception, of why Alice in Wonderland syndrome is called a syndrome in the first place and why it is not included in major classifications such as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and the *International Classification of Diseases* (ICD) . Finally, throughout this book, I will seek to shed light on the issue regarding the relationship between this enigmatic group of perceptual phenomena, the *Alice* story, and the possibility that Charles Dodgson may have drawn on personal experiences to describe them. Although far from definitive, I hope that the answers will enhance our understanding of this orphan disease ; that it may help to bring it to the attention of patients, their loved ones and those looking after their health; and that it may inspire researchers to delve deeper into the many mysteries that remain.

References

1. Blom JD (2016) Alice in Wonderland syndrome. A systematic review. *Neurol Clin Pract* 6:1–12 [[Crossref](#)]
2. Blom JD (2017) Alice in Wonderland syndrome. In: Sharpless BA (ed) *Unusual and rare psychological disorders. A handbook for clinical practice and research*. Oxford University Press, Oxford, pp 265–287
3. Carroll L (1987) *Alice’s adventures in Wonderland and Through the looking-glass*. Chancellor Press, London
4. Burton T (2010) *Alice in Wonderland*. Walt Disney Pictures, Burbank
5. Elfman D (2010) *Alice in Wonderland OST*. Walt Disney Records, Burbank
6. Frederiks JAM (1963) Macrosomatognosia and microsomatognosia. *Psychiatr Neurol Neurochir* 66:531–536 [[PubMed](#)]
7. Lynch DK, Livingston W (1995) *Color and light in nature*. Cambridge University Press,

Cambridge

8. Rovelli C (2018) *Het mysterie van de tijd*. Translated by Boeke Y, Krone P. Prometheus, Amsterdam
 9. Lovett CC (2005) *Lewis Carroll among his books. A descriptive catalogue of the private library of Charles L. Dodgson*. McFarland & Co., London
 10. Lindseth JA (2015) *Alice in a world of Wonderlands: the translations of Lewis Carroll's masterpiece*. Oak Knoll Press, New Castle
 11. Kundera M (1984) *The unbearable lightness of being*. Translated by Heim MH. Harper & Row, New York
 12. Frost M, Lynch D (1990) *Twin peaks*. Paramount Pictures, Hollywood
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Footnotes

¹ Of course, Artemis is not the real name of my patient. Throughout this book, all the patients' names are fictitious; however, their stories are not.

² *Alice in Wonderland syndrome*. From Wikipedia, the free encyclopaedia. Retrieved on February 16, 2016, from https://en.wikipedia.org/wiki/Alice_in_Wonderland_syndrome

³ This phenomenon is known as Buddha's halo, a physical illusion consisting of multi-coloured rings of light that can be seen around the shadow of one's head when one is looking at a bank of clouds opposite a low-positioned sun behind one's back. For more information, see *Color and Light in Nature* by Lynch and Livingston [7].

⁴ I remember telling Renate that now I finally understood the line in Pink Floyd's *Comfortably Numb*, where they sing, 'When I was a child, I had a fever—My hands felt just like two balloons'.

2. Inside the Consulting Room

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

When I was about to become a psychiatrist, I was invited for an assessment interview by the director of the department where I had just finished the final stretch of my training programme. Despite being 15 years my senior, he had a youthful and athletic appearance, which may or may not have been due to the fact that, at middle age, he was still running the annual marathon. Perhaps fitting for a runner, he was a man of few words. Also, he used to take his time searching for the right words to speak—so much so, that it was sometimes hard for us residents not to say out loud what we thought he was going to say. Luckily, one of the basic skills of the psychiatric training programme involved the art of biting our tongue and waiting for whatever our conversational partner would bring up next. That skill came in handy while dealing with the director and saved me, at least, from a great deal of social awkwardness during my training.

Because of his manner of speech and perhaps also because of the way he could silently observe you with his blue, bespectacled eyes, you never quite knew what to expect from him. However, his words were always spot on and worth the wait. He had great clinical insight and, in the past, he had often surprised me with his extensive knowledge of the history of psychiatry. So there I was, seated at the desk of this silent and uncannily knowledgeable director, ready to receive his verdict on the way I had been doing my work over the last year of my residency training. By then, having got to know something of his ways, I did not expect any abundant praise—

that was simply not his style. Nevertheless, I was somewhat disappointed when he ticked off the obligatory administrative issues on his checklist in utter silence and then concluded our meeting with the enigmatic remark, ‘Well, Jan Dirk,’ (silence), ‘Well, I think,’ (silence), ‘I think that I would certainly trust you...’ (silence), ‘...with a patient.’

That was it. The director considered me worthy of being trusted with a patient. Over dinner that night, I told Renate about the assessment interview; she shrugged and said he would probably have had a bad day or something like that. I went to the kitchen to pile up the dishes, filed the incident away as one of those quirky experiences a resident has to go through, and never gave it another thought. That is, until years later, the director contacted me with the request to take under my care his youngest son, Dimitri.

2.1 The Man Who Sensed the World to Be Alive

So that is how I got to meet Dimitri, a good-looking young man in a beany hat who had been diagnosed with schizophrenia . He had been put on clozapine , an atypical antipsychotic that is usually reserved for therapy-resistant patients, which implied that he must have tried at least two other types of antipsychotic medication in the past. However, he did not show any of the usual side effects. Similar to his father, he had the lean build of a runner and moved around with enviable grace. It made me doubt whether he actually took his medication—but his plasma levels proved to be within the therapeutic range. So, I was not surprised to learn that Dimitri actually was a runner and that he was also into boxing, inline skating, cycling and rock climbing. Also, like his father, he was very smart. He had been studying philosophy but, during his freshman year, he had to stop because of severe paranoia. He was admitted to a university hospital (under the Dutch equivalent of the Mental Health Act), where he had been treated with the classic antipsychotic, haloperidol . Nonetheless, his paranoid state had led to various aggressive outbursts in the hospital and at home. He was admitted a second time and, following his discharge, had come under the care of a psychiatrist elsewhere. In retrospect, his illness had probably begun more than 10 years earlier, with signs of what appeared to be indicative of attention-deficit hyperactivity disorder (ADHD) . Aggravated by the abundant use of cannabis , as well as the use of methylphenidate (an

amphetamine used to treat ADHD) in dosages that far exceeded that which had been prescribed (which perhaps also led to sleep deprivation), Dimitri had crossed the line to frank psychosis.

I felt for him.

And I felt for his father.

After all those years, I finally got an idea of what might have been going through his head during that assessment interview; in the meantime, having become a father myself, I could only empathise with the grief he must have felt all those years in the face of his son's journey through life. Having a psychotic son or daughter is something incomparably sorrowful, with ramifications for all family members. Under such circumstances, a father who happens to be a psychiatrist may have the advantage of knowing what it is and what to do; however, that advantage is outweighed by his knowledge of the myriad sad scenarios that may be lying in wait. Realising that, I no longer envied the scope of my former director's knowledge.

The reasons why Dimitri had had himself referred to me were threefold—in fact he had it all written down on a crumpled sheet of paper that he pulled from his rucksack. In the first place, he wanted to be able to resume his philosophy study. That was something I could relate to and wanted to do my best for. On the other hand, I knew that my chances to make that happen were somewhat limited, since psychosis and academic achievement generally do not go well together. There are exceptions, of course, as exemplified by the mathematician and Nobel Prize laureate John Nash (1928–2015), although anyone who has read Sylvia Nasar's biography of Nash [1] or seen *A Beautiful Mind*, the movie based on Nash's life [2], will realise that it takes almost a miracle to live up to the demands of university life when the mind transforms the world-as-we-know-it into something entirely different.

Secondly, Dimitri wanted to know whether he might be suffering from 'ADD psychosis', a rare combination of ADHD and psychotic disorder . This was something his father had suggested to Dimitri's former psychiatrist and a subject I had published on with Sandra Kooij, an internationally renowned expert on ADHD [3]. Although both Dimitri's father and I knew that this should be explored, we also knew that this might entail a rather tricky treatment regimen of clozapine *and* methylphenidate . This combination is vehemently discouraged in the literature, and very few psychiatrists are willing to try it with their patients. This is, of course, not

without reason as methylphenidate has the potential to promote psychotic symptoms and, in Dimitri's case, had almost certainly contributed to his first psychosis . Nevertheless, this was an avenue I was willing to explore but with very great caution.

In the third place, despite his relative stability on clozapine , Dimitri suffered from various perceptual symptoms that he hoped I could help him with.

Like his father, he was thoughtful and soft spoken. Unlike his father, however, he was reluctant to sit still and remain in contact with me for more than a few minutes at a time, making it difficult to hear him out on the particulars of his complaints. Thus, it was only over the course of various sessions that I learned that he experienced the inanimate world as being somehow 'alive', although I was never really sure whether I had fully grasped what he meant by that.

As Dimitri had initially confirmed my suggestion that he sometimes saw movement in smooth surfaces (such as the walls and floors), as if a ripple or a wave were going through them (a type of metamorphopsia known as *kinetopsia*), I took that for granted and merely referred to it occasionally when we assessed his progress on various types of medication. 'Are you still seeing movement in the walls, Dimitri?' I would ask—and he would answer that things were a little better now, or still the same. It was many years later that he told me that I had never properly understood him and that he had simply said 'yes' to my suggestion back then because he could not stand all the questioning on my part. Although I know I can be persistent when it comes to asking about the specifics of perceptual phenomena I can, apparently, also go too far. I was sorry to hear that—because I had been genuinely interested in whatever it was that he was perceiving.

He then told me that it was more like a kind of knowledge, or intuition, that the world around him was alive. It was a sensation that he associated with the Japanese anime *Arrietty* , in which tiny people called 'Borrowers' populate a house without the human inhabitants knowing about it [4]. Not that Dimitri ever saw any Borrowers, or even thought that they were around. It was more like a hunch that someone or something *could* be out there, a form of sensed presence perhaps. Later on Dimitri compared this sensation with something he had read in a book by Rudolf Steiner (1861–1925), the founder of anthroposophy , who had claimed that he was able to

see whether something was alive or not by examining its aura . Although Dimitri did not think that he saw any auras, he did see purple-coloured patches. He described them as shapeless forms which moved along with him whenever he walked the street or rode his bicycle, and which seemed to attach themselves to cars and facades and other objects as he passed them by. In addition, he told me about brightly coloured lines that he saw around objects, like an extra contour. He said that the sheets of paper lying in front of us on the table had lines around them that were a bright, greenish yellow—something I myself definitely did not see.

I decided that these perceptual distortions were something I wanted to focus on first, together with Dimitri's ambition to get back to university. It was uncertain whether the latter aim was even feasible but, if it might be, I really wanted to help him get there and, if not, it would be best to know as soon as possible and then help him find a more suitable occupation. Dimitri's parents had always helped him in any way possible, given the circumstances, to make the best of his talents. Thus, over the years, his father went running and rock climbing with him, took him on camping trips to the Wadden Islands, and on bicycle trips and other sports travels. Both his parents stimulated his reading and his physical activities, helped him to find a sheltered home and did numerous other things for him. My role became to explore with Dimitri the sensitive issue of ambition versus capacities with respect to studying: we discussed the books he had been reading, traded ideas on philosophy (but also on fantasy novels and movies, which he seemed to like a lot more) and, one day (on Dimitri's request), I accompanied him to a pre-university studying facility in Leiden where he hoped to get back in shape intellectually.

Another reason why I decided to target the perceptual distortions first, was that they had struck me as atypical for what psychiatrists call 'schizophrenia .' Not that any two persons with this diagnosis ever have the exact same symptoms, but about 70% of them are constantly plagued by voices whereas visual phenomena (if present) tend to be transient in nature. Besides, I was not eager to go down the ADD-psychosis road, as I already had an idea of how the methylphenidate might have contributed to Dimitri's transition to psychosis .

So, the visual distortions it had to be, even though it would not be easy to pin down the exact nature of the purple-coloured patches. Positive disorders of visual perception come in many shapes and varieties, and their

phenomenological features have been well documented. Generally, the more specific those features are, the easier it is to identify them and to infer the brain mechanisms responsible for their mediation. However, Dimitri's patches were relatively uncharacteristic in nature. They could be anything ranging from negative or even positive afterimages to coloropsia, which is the perception of hallucinated colours produced more or less at random by the brain. Alternatively, as they apparently attached themselves to objects in the street, they might also be a type of metamorphopsia called *illusory visual spread*. Illusory visual spread is characterised by the presence of certain colours or patterns in the outside world, which—as experienced by the observer—spread out to other objects so as to make them look similarly coloured or patterned. In their paper on positive pathologies of vision, Dominic ffytche and Robert Howard (from London's Institute of Psychiatry) illustrate this phenomenon by means of a drawing of a couch and various armchairs with small plaids on top of them, and a second drawing, in which the pattern of the plaids has spread out to cover a large part of one armchair, plus the whole couch [5]. Perhaps that, I thought, provided the best match with Dimitri's purple-coloured patches, although I was not completely sure whether they were the same phenomenon.

By comparison, the brightly coloured contours were easy to identify. They are known as a corona phenomenon, yet another type of metamorphopsia [6] which, in the older literature, is also called border, shiny ring and halo.¹ Although its exact cause is yet to be determined, we know that it is produced by the brain, probably by cell columns in visual cortex that have a function in detecting contours or border areas between differently coloured objects [7].

As is the case for other types of metamorphopsia, structural or functional aberrations can sometimes be demonstrated in the brain with the aid of neuroimaging techniques. I discussed this with Dimitri and his father and we agreed that a brain MRI and an EEG should be obtained first. The MRI and EEG showed no abnormalities, and neither did any of the blood tests I had ordered. When the three of us met again, we discussed how to proceed. There are no evidence-based guidelines for treating metamorphopsias in the absence of any demonstrable pathology; however, as the various types of antipsychotic medication had had no effect whatsoever on Dimitri's metamorphopsias in the past, the logical thing was to try something different. Therefore, I proposed to add the antiepileptic

drug, valproic acid, in the hope of neutralising any epileptiform activity that could theoretically still be present in Dimitri's brain, even though no indication of it had shown up on the EEG.² I reasoned that structural brain damage, however subtle it might be, had been sufficiently ruled out by the MRI scan. Moreover, a structural cause would more likely have entailed a constant presence of his perceptual symptoms, whereas Dimitri's were intermittent in nature. I took this to indicate a possible functional cause, implying that the brain's function was impaired, even though its overall anatomy was apparently intact. Dimitri and his father understood that adding the valproic acid would involve an experimental type of treatment. Nevertheless, knowing that this medicine is also used sometimes to potentiate the antipsychotic effects of clozapine in patients with therapy-resistant psychosis, they both consented.

When Dimitri came to visit me 2 weeks later, he told me that he was experiencing substantially less movement in the walls. Also, although the coloured lines and purple patches were still there, they were not as frequent and were at a much lower intensity. At that point, he had not told me that he did not actually see any movement at all in the walls, so I had nothing better to say than that these first results were promising. Knowing that the initial response to any type of treatment in psychiatry tends to be favourable (perhaps due to a placebo effect, or to the patient's unconscious wish to please the therapist), I had doubts as to whether this would last very long. Nonetheless, at least there were no side effects and, all things considered, it was not a bad start. I told Dimitri that a higher dose might yield even better results and, with his consent, doubled the dose.

A few months went by during which I saw Dimitri at intervals of every 2 or 3 weeks. Each time he reported on his perceptual symptoms; sometimes they had receded further into the background, and sometimes they had remained the same. However, when one day I proposed a further dose increase, Dimitri was reluctant to comply. When I asked him why (especially as he had never experienced any side effects), he explained that the metamorphosias, although still present, no longer really bothered him. Looking back, he doubted whether the valproic acid had had anything to do with this. The explanation about their neurobiological basis had, apparently, proved sufficiently reassuring to stop him worrying. Moreover, he seemed to somehow cherish the idea of the world being 'alive' and did not seem ready to have that taken away from him.

Meanwhile, I realised that, during all those months, Dimitri had not shown any signs of the paranoid psychosis he had apparently suffered from in the past. Since antipsychotics, due to their ability to lower the threshold for epileptic activity, also have the potential to *cause* metamorphopsias and other visual symptoms [8], I thought we should try to decrease the dose of clozapine and see whether that might yield beneficial effects. To many patients, that idea would have been music to their ears—but not to Dimitri. He was well aware of the stability that the clozapine had brought into his life and he was too perceptive to put that at stake. Therefore, at first, we kept the dose of his clozapine unaltered. By this time, the two of us had developed a good working relationship and I trusted that he would tell me whenever he was ready to decrease the dose.

That moment came about 2 months later.

We sat down to anticipate the possible effects of a lower dose on his metamorphopsias. We also discussed what could happen if his former paranoia might inadvertently return and compiled an inventory of warning signs he should be aware of. Despite these precautions, however, and despite the fact that I lowered the dose by only a fraction, Dimitri decompensated within a few weeks and had to be admitted for a third time. He was severely paranoid and agitated and, in that state, I got to see a completely different side of him.

As long as I had been treating him, I had often wondered whether his first psychosis might have been a simple reaction to the metamorphopsias, and whether these perceptual symptoms might have provided the trigger for any paranoid ideas that followed afterwards. After all, seeing coloured lines and patches that apparently no one else can see, and experiencing the world as being alive, is bound to make one wonder what is going on and send one searching for explanations—even if that requires considering unconventional possibilities. However, seeing him now and hearing him speak of his alleged ties with the Dutch royal family, his powers to influence the weather and many other delusional ideas, I could only conclude that he had been correctly diagnosed with psychotic disorder in the past. Dimitri stayed for a week in our secluded nursing ward, during which time he remained agitated and confused but, fortunately, accepted a return to his former dose of clozapine. Upon his specific request, he was then discharged. At home, it took him 3 whole months to regain his prior level of functioning. Throughout that period, he continued to see me in the

outpatient clinic at more or less regular intervals, although sometimes he skipped our appointments for reasons that were unclear to me. Even though we later tried out various different types of medication in addition to the clozapine, his visual symptoms remained basically unaltered.

Over the years, Dimitri's life also remained basically unaltered. He never succeeded in getting back to university and, instead, kept reading his fantasy novels, together with the occasional book on philosophy. He often told me that he would like to live on his own and, eventually, he did move to another place—albeit within the sheltered-living circuit. Also, although he kept up his stiff training schedule, he often complained to me that he could have attained so much more if he had quit the cannabis and other substances that he continued to use. Apart from the cannabis, he also got hooked on cocaine, which made his paranoid psychosis flare up now and again, leading to agitated discussions with his neighbours and carers. Fortunately, it never got so out of hand that he needed to be admitted again. On the positive side, Dimitri got himself a job as a freelance bike courier, thereby earning a living doing one of the things that he liked most in life and was certainly good at. As for his father, I don't think that he ever came to full closure with his son's life. Nevertheless, I assume he must have realised that the saddest of scenarios had not played out and he must have remained thankful for every opportunity to share time with his son and to see him gradually finding his way through life.

Looking back at Dimitri's story, you may wonder why I decided to focus on his visual symptoms when there were so many other problems (e.g. psychopathology, substance abuse, independent living) that dominated his life. I wouldn't be surprised if you considered these visual symptoms to be the least of his problems, even though Dimitri himself had singled them out as one of the three things that he wanted help with when he first came to see me. It is always a matter of careful consideration, for both doctor and patient, to determine what is important, what is less important and what should be treated first. In order to explore the impact of such symptoms when they present in total isolation, let us now meet Paul, who suffered from a single, peculiar distortion called plagiopsia that brought him to such despair, that he was on the verge of giving up life altogether.

2.2 The Boy Who Saw Everything as Slanted

Paul was a young boy aged 6 years. I first heard of him at the inaugural ceremony of my friend, Bert van Hemert, who had just been appointed Chair of the Psychiatry Department at Leiden University. Bert had been a university professor for 3 years, and this was his second Chair. Since I had the honour of acting as his master of ceremony, I got to meet all of his guests. The way these things go, I made announcements at designated moments, answered questions, showed people where to hang their coats or find the restroom, and indulged in a lot more small talk than I am accustomed to. Prior to the ceremony (held at Leiden University's exquisite 16th-Century Academy Building), we were sitting in the wood-panelled room behind the Grand Auditorium greeting close friends and members of Bert's extended family, chatting away about this and that, and raising our glasses in his honour. One of the guests, a man in his 40s who had introduced himself as Jean-Eric, asked me what I did—I told him that, like Bert, I was a psychiatrist and that I worked at Parnassia Psychiatric Institute. He wanted to know what kind of a psychiatrist I was and I told him that I was primarily concerned with psychotic disorders, notably hallucinations.

When people ask me additional questions beyond that point, they run the risk of being exposed to all kinds of nerdy stuff about my work and research. As Jean-Eric ventured to go on, before I knew it I found myself sketching a rudimentary brain on a napkin and pointing out cortical areas and networks that play a role in perception, meanwhile updating him on neurobiological hypotheses, both old and new. Inevitably, perhaps, I also mentioned Alice in Wonderland syndrome. To my surprise Jean-Eric was eager to know more, and after I had described a number of symptoms of Alice in Wonderland syndrome, he deadpanned: 'I know someone who has this'.

I remember that, at first, I did not really believe him. At that time, I had not personally encountered many people with Alice in Wonderland syndrome and, moreover, whenever I gave lectures on the subject I tended to get questions about related phenomena, rather than the ones that I was actually talking about. However, when Jean-Eric went on to describe a boy who was seeing everything as slanted, it dawned on me that we were indeed talking about the same thing.

Paul was the son of some friends of Jean-Eric. The boy's parents, worried sick, had taken him to various specialists, but none of them had been able to tell them what their son was suffering from. I pointed out that I

specialised in adult psychiatry with little knowledge of paediatric pathology, but offered to meet Paul to see what I could do. The next day I received an email from Mrs. Brevis, Paul's mother, and a week later, on a bright and sunny day, the two of them walked into my consulting room.

At first sight, nothing seemed to be the matter with Paul. He was a healthy-looking Dutch boy with flaxen hair, the type I used to see in dozens when I picked up my children from school in the afternoons. He walked with a steady gait, shook my hand politely and sat on the chair I offered him. I was surprised to find that he was not at all intimidated by meeting a stranger in a suit who cross-examined him for over an hour; on the other hand, I was hardly the first doctor he had ever met. He answered most of the questions himself, carefully choosing his words and expressing himself with precocious precision; the remaining questions were answered by his mother.

Paul's story went like this.

During the summer vacation of the previous year, he and his family had spent several weeks camping in France. During the long drive back to Holland, Paul had fallen asleep in the back seat of the car. When they stopped at a roadside restaurant, he had awoken and, while stepping out of the car, had noticed that everything looked slanted. Lampposts, trees, buildings—they were all off balance, tilting to one side. He told his parents about this and they had thought that he had slept too deeply or had a stiff neck from lying in a crooked position—in the full car, there was little room for him to lie down comfortably. They went into the restaurant, had a quick bite amid the clamour of truckers, tourists and other travellers, went back to the car and drove home.

The next morning, Paul woke up in his own bed and found that nothing had changed. Everything still looked slanted and, again, he complained about this in front of his parents. He had difficulty walking and it was particularly hard for him to participate in physical games and sports activities. His parents took him to the family physician who examined Paul but did not find anything out of the ordinary. Following the parents' lead that he had slept all cramped up during the long journey home, he referred him to a physiotherapist to have his neck and back examined. In addition, the family physician sent him to the hospital to have a neck X-ray; again, nothing out of the ordinary was found. By the time the results came back

one week had passed and Paul's condition had remained unaltered. The family physician then decided to refer him to an ophthalmologist.

Ophthalmologists are familiar with metamorphopsias . Whenever a person reports that straight lines look wavy (something like the grout joints on the bottom of a swimming pool), they immediately examine both retinas for signs of ablation . Retinal ablation (or retinal detachment) is a condition in which the light-sensitive layer of cells inside the eyeball peels away from the underlying layer that nourishes it. Deprived of blood and, thus, of oxygen the retinal cells die, causing loss of vision in the process and, occasionally (depending on the extent of tissue damage) total blindness of the diseased eye. Therefore, *ablatio retinae* is one of the most time-critical conditions in ophthalmology. What is needed in such cases is an emergency surgical procedure or (when the detached area is relatively small) emergency treatment with the aid of laser surgery or freeze treatment (cryopexy).

Although, technically, the seeing of wavy lines is called dysmorphopsia , many ophthalmologists refer to it by the generic term 'metamorphopsia'. So, when I said that ophthalmologists are familiar with metamorphopsias, what I meant is that they are familiar with dysmorphopsias, whereas other types of metamorphopsia (most of which are caused by cerebral rather than ocular pathologies) tend to lie outside their field of expertise.

This was also the case with Paul's ophthalmologist. He carefully examined the boy's eyes and visual acuity , gave him the reassurance that there was nothing wrong, and referred him back to the family physician. Meanwhile, Paul's condition had not improved. It was now several weeks since he had returned from France, he was still seeing everything as slanted and was tiring of the idea that nothing could be done about it.

His family physician then referred him to a paediatrician, who also examined him thoroughly, searched for any signs of prior infection and ordered blood tests to rule out that possibility. In addition, he arranged an EEG and a brain MRI . As none of the tests were positive, the paediatrician also had nothing to offer. Paul found it hard to accept that this condition might stay with him forever and, fearing this, became depressed. At some point, he even told his parents that he would rather die than live with this situation for the rest of his life.

Needless to say, their hearts broke.

So there they were, mother and son, seated at my table in the sunlit consulting room, after a year of uncertainty and agonising worries. Fortunately, by this time, Paul was experiencing increasingly longer intervals each day during which he was able to see vertical lines the way they were supposed to be. However, several times a day he still saw them as slanted—sometimes to the left, and sometimes to the right. When asked about it, he made it clear to me that he had never experienced any other visual distortions . I sat with him and his mother behind my computer, closed the blinds so that we could clearly see what was on the screen, and showed them a PowerPoint presentation with simulations of various types of metamorphopsia that I use for lectures. With the exception of the one in which a building is seen keeling over to the right, Paul did not recognise any of the phenomena on display. Nor had he ever experienced any other symptoms of Alice in Wonderland syndrome, such as somesthetic distortions or time distortions . Since all the necessary auxiliary investigations had already been carried out, all that remained for me to do was explain to Paul and his mother what I thought that he was suffering from.

I told them that the technical term for seeing everything as slanted is *plagiopsia* and that the phenomenon is considered a type of metamorphopsia , which, in turn, is considered a symptom of Alice in Wonderland syndrome. I showed them a picture of the brain, pointed out the occipital area with its function in visual perception and explained that, throughout a layer called V3 , there are stacks of nerve cells called orientation columns , which respond to vertical lines, oblique lines, horizontal lines, concentric circles and so on, whenever such elements are present in the visual input picture. I told them that what we see in ordinary visual perception is the net result of what these cell columns (and many, many other columns, as well as larger neuron populations) tell us about what is present in the visual input picture. When they inform us correctly about the presence of vertical lines, colours, movement and so on, then we are able to perceive properly what is going on in the scene that we are looking at. However, when one of those neuron populations malfunctions and tells us that something is there that isn't there or, alternatively, fails to register something that is actually there, we get a condition such as the one Paul had been suffering from. In his case, my guess was that the visual cell column tasked with registering the presence of vertical lines was

malfunctioning, and that perhaps neighbouring cell columns (with their function in registering the presence of oblique lines) had begun to show spurious activity. The result of this was that Paul failed to see vertical lines as vertical and, instead, perceived them as slanted either to the left or to the right.

The question was, of course, how it was possible that this finely tuned mechanism had suddenly gone off the rails while he was travelling back from France and, even more importantly, what could be done to get it working properly again. To the first question, I had no answer. As my fellow specialists had already thoroughly ruled out the most likely causes of plagiopsia with all their testing, I had no illusions about the chance of uncovering its genesis with additional testing. Besides, it was highly unlikely that any damage to a single-cell column—if present—would be detectable on a brain MRI or EEG .

In children, the most frequent cause of long-lasting metamorphopsias is encephalitis , an infection (mostly viral in nature) of the brain. In the literature, Epstein–Barr virus infection is mentioned as the most common cause among the encephalitis-induced metamorphopsias. Until now, a lumbar puncture had not been performed; however, as Paul had not shown any fever or other clinical signs of encephalitis and the blood tests were clean, that possibility seemed unlikely. In adults, the most frequent cause of metamorphopsias is migraine . Although migraine is chiefly known as a disorder characterised by severe headaches , it can also be present in the absence of any headaches. In such cases, it is appropriately called ‘migraine without headache’ . However, Paul had never experienced any migrainous headaches and, if it had been a migraine without headache, any accompanying visual symptoms would have been episodic in nature, similar to the way that full-blown migraines tend to come in the form of ‘attacks’. So, migraine also failed to qualify as a likely causal mechanism. Although many other known causes of metamorphopsia exist, none of them seemed relevant in Paul’s case. Thus, the cause of his chronic plagiopsia remained a mystery to us and, I too, had come up empty-handed in that respect.

To answer the question concerning what could be done about it, I offered a summary of the literature on metamorphopsias in children; this states that, in approximately half of the cases, if no underlying cause can be proven to exist, the symptoms vanish spontaneously within 1 or 2 years. I

also offered the possibility of an experimental treatment with valproic acid , the same antiepileptic I had prescribed to Dimitri.

Mrs. Brevis thanked me for my explanation and promised that she would think about the treatment proposal for her son, although she added that she was inclined to spare Paul the burden of having to take any pills, especially when there was a chance of him getting better without them. When we spoke on the phone a week later, she had made up her mind about it. She told me that Paul had been sufficiently reassured by my analysis of the situation and that she and her husband preferred to give it a try without any medication. They would wait and see whether the symptom-free intervals continued to increase in length and would come back to me if they did not. That is how we parted; when I emailed Mrs. Brevis one year later, she wrote back to say that Paul was doing very well and that the plagiopsia had indeed disappeared without a trace. When one of my residents, Vivian Buijs (now a psychiatrist) approached Mrs. Brevis 3 years later in the context of a follow-up study, she was kind enough to answer all of her questions. However, she did not want Paul to answer them in person as, by that time, he had largely forgotten the whole episode and she did not want him to be reminded of it. Vivian and I totally understood this and did not want to bother him either.

Fortunately for Paul, he had suffered from this particular type of metamorphopsia for only 2 years—however, what a burden it had been for him and his parents and how long those 2 years had seemed. On the outside, nothing appeared to be wrong with him—no broken leg, no baldness or wasting away due to some aggressive cancer treatment, nothing like that. And yet he had been on the brink of giving everything up, at the age of just 6 years. Paul’s case aptly demonstrates how disrupting a single, isolated type of visual distortion can be. If this makes you wonder what it might be like to experience one such symptom for an entire lifetime—let us see how Mrs. van Nuys dealt with prosopometamorphopsia .

2.3 The Woman Who Saw Dragons

In July 2011, I received an email message from Mrs. van Nuys. It was a brief, simple message in Dutch which translates as follows:

Hello Dr. Blom,

Please read my e-mail correspondence below. Perhaps you will be my salvation.

*With kind regards,
Sophia van Nuys*

Since I was not acquainted with any Mrs. van Nuys, I had no idea what this was all about; so, as requested, I scrolled down to look at the correspondence that she referred to.

The message below her short e-mail was from Kate Edgar , assistant to the neurologist Oliver Sacks (from New York), who thanked Mrs. van Nuys on his behalf for contacting him and apologised on his behalf for not being able to diagnose her from a distance. As an alternative, she wrote, Professor Sacks had recommended me.³ My e-mail address had been included and, apparently, that was how Mrs. van Nuys had found me. Underneath Ms. Edgar's message was the original message that Mrs. van Nuys had sent to Sacks. In it, she explained that she was seeing all kinds of monsters, that no specialist appeared capable of helping her, that her neurologist had brought the work of Professor Sacks to her attention and that she hoped he would be able to tell her what she was suffering from, as her own neurologist had suggested that 'hers was a unique academic case'.

When I replied and thanked her for her e-mail message I also clearly explained in advance that I was not Oliver Sacks and, therefore, was not sure whether I could be of any help. Nonetheless, she said she wanted to come to The Hague for a consultation.

When Mrs. van Nuys arrived a few weeks later, she was not at all what I had expected. She was a lively, light-hearted Asian woman with close-cropped hair who strode towards me from the waiting room. She was 52 years old, although she could easily have passed for 30-something and behaved accordingly. I led the way upstairs to my consulting room and, as soon as we had sat down and exchanged the necessary formalities, she told me the following story:

Mrs. van Nuys saw dragons—or rather, dragon faces. As far as she knew, she had always done so. From her earliest childhood onwards, whenever she looked at another person, she first saw a regular human face and then, after several minutes, she could not help but watch it morph into something different. As she explained to me: the person's face would turn black, beginning at the sides and then all the way to the middle, until it was

evenly black, displaying a reptiloid aspect of the skin. Meanwhile, a snout would appear, as well as a pair of long, pointy ears. The eyes would take on a bright, shiny aspect and were coloured red, yellow, green or blue. When the transformation was complete, she would be staring into a black dragon's face with fierce eyes and, for the remainder of her time in that person's company, that was what she would see.

Like the rest of us, as a child, she had simply lived with the way she had learned to perceive the world and, for a very long time, she had had no idea that this was *not* how her family members, neighbours, friends, classmates and in fact anyone else, were seeing each other's faces. It took her until early adolescence to find that out, and when she finally did, it hit her like a ton of bricks. She felt lonely and isolated and, understandably, became depressed. She took to drinking and found out that alcohol helped her cope with these monstrous faces and, in the process, allowed her to be more sociable. Looking back, she had begun to realise that she had always had difficulty reading other people's faces and, thus, of keeping track of any nonverbal clues during conversations.

We may not always be aware of this but, in everyday life, we constantly check each other's faces for signs of approval or dissent, reassuring ourselves that the story we are telling is still appreciated, finding out whether people around us are getting bored or not, verifying whether they still like us or not, probing as to whether they have good or bad intentions and so on. Subconsciously, we are even able to read the micro-expression s that glide over people's faces—the beginning of a smirk, which betrays their secret joy over something that we totally blew; the fraction of a second during which we get a hint of some repressed emotion; the quick glance that betrays what they are really thinking. Such micro-expression s typically last a 25th of a second, which is too brief for us to register in a conscious way. Nevertheless, we all depend on those ultrafast giveaways to survive socially and, as Mrs. van Nuys had discovered the hard way, not being able to read them is a serious disadvantage in life.

Consequently, she had repeatedly experienced failures of communication and, in the consumption of alcohol , had found a compensatory mechanism that enabled her to deal more appropriately with social situations—she was able to better tune into others and felt less inhibited in their presence. The downside of this solution was that she developed a serious alcohol problem, dropped out of high school and did

more-or-less nothing for many years—while everyone in her vicinity went to college, found a job, got married and, the way things go, did all they could do to make the best of their lives. During her 20s, she met the man who was now her husband. She then managed to quit the alcohol, finished her education, found herself a job as a school administrator and gave birth to a daughter. However, due to her daily recurring visual symptoms and the ensuing problems with other people, she continued to have many conflicts. Whereas her husband and daughter knew how to deal with that, at work she had a hard time explaining what she was suffering from and why she had such difficulty reacting to social cues, even with colleagues who were willing to hear her out. No one had ever heard of the curious condition she suffered from. Being unable to come up with a diagnosis, or to even explain what her symptoms were called, she was met with doubt and disbelief and, due to unresolved misunderstandings, she was repeatedly forced to change jobs.

By the time Mrs. van Nuys came to see me, she was still seeing people's faces change into dragon faces—tens to even hundreds of times a day. In addition, she often saw similar dragon faces emerge from her computer screen or drift towards her out of a wall, or a wall socket; moreover, at night, when she awoke, the room could be filled with numerous copies of dragon faces staring back at her from out of the darkness. In contrast to Paul, she could not remember ever having been symptom-free, not even in early childhood.

Similar to what I had done with Paul, I showed her the PowerPoint presentation with simulations of metamorphopsias . She recognised none of them but was mildly shocked when I showed her an image of a man with a lion's face. When we were finished, she told me that she had never experienced any other types of distortion but added that, instead of that, she sometimes saw giant ants crawling over her hands—not over the table next to her hands, not over her arms but only over her hands.

As she was of Asian descent, I asked her whether the dragon faces had ever reminded her of the dragon masks characteristic of her culture. She said that she had never given it a thought but had no associations of that kind. On further questioning, it struck me that she had no associations whatsoever with the dragon faces. They frightened her, as they had done all her life and, despite her cheerful appearance, she still felt sad and depressed because of all the havoc those faces had wreaked upon her. And yet, in

contrast to Dimitri, she had never developed any delusional ideas. In fact, she had managed to cope admirably with this neurobiological curse and had not succumbed to psychosis; that feat alone was testimony to her incredible strength. Moreover, the way I understood it, finding the man she had married (whom I met during a subsequent visit to the outpatient clinic) was the best thing that had ever happened to her. If anyone qualified as being her ‘salvation’, it was him.

Like Paul, Mrs. van Nuys had previously consulted various specialists, including a psychiatrist and a neurologist. The psychiatrist had attempted to treat her with antidepressants and antipsychotics, both of no avail. Her medical history included a caul birth, recurring instances of sensed presence, recurring headaches (of a non-migrainous nature), and recurring urinary tract infections. My psychiatric examination revealed nothing out of the ordinary, except for the dragon faces, the hallucinated ants and an underlying depressed mood (which, considering the circumstances, was not really surprising). Mrs. van Nuys showed proper insight into her situation. She guessed that the dragon faces were probably due to some neurological disorder, which might or might not have something to do with her being born with a caul over her face. I agreed with that, although I had to confess that I had never heard of a case like hers before. I told her that the only comparison I had was a patient who, while switching from one type of antipsychotic to another, had temporarily experienced everyone’s faces as displaying huge, prominent eyebrows. Although this had really frightened my patient, I had been able to reassure her that this was probably due to the medication switch—which it most likely was, as this peculiar symptom vanished completely within a few days. At that time, I had carried out a literature search to see whether this symptom had been described before and had found a handful of publications on the perception of distorted faces, called *prosopometamorphopsia* (from the Greek word *prosopon*, which means face, and *metamorphopsia*).

So that was what I told Mrs. van Nuys: that she suffered from *prosopometamorphopsia*, a type of *metamorphopsia* which is, in turn, considered a symptom of Alice in Wonderland syndrome. The dragon faces that she saw when she was not in anybody’s company—and, of course, the ants she occasionally saw on her hands—were visual hallucinations. However, given the similarities with the *prosopometamorphopsias* (visual, animal nature, movement), my guess was that they stemmed from the same

neurobiological source or sources. I explained that we would have to carry out auxiliary investigations to see whether there were any signs of underlying pathology of the brain and that, at this point, I could only tell her that metamorphopsias were rare, with this specific type being so rare that I was not aware of anyone having ever published on this before. Even though I could not offer her anything more at that time, Mrs. van Nuys told me that she was glad that her condition now had a name. She wrote down the term ‘prosopometamorphopsia’ and resolved to search the Internet for any additional information. I also gave her a copy of a paper our group had published on a patient with different symptoms of Alice in Wonderland syndrome, which we had treated with the aid of repetitive transcranial magnetic stimulation [10]. Mrs. van Nuys concluded her visit by saying that she appreciated the term ‘Alice in Wonderland syndrome’, as she herself had more than once felt like Alice, all alone, in a really weird and curious world.

With Mrs. van Nuys’ consent, I conferred via email with Oliver Sacks and also with Dominic ffytche, a leading international expert on visual hallucinations in the UK. Neither of them had ever encountered a patient like Mrs. van Nuys, nor were they aware of any descriptions of comparable cases. As we later wrote in an article on the case of Mrs. van Nuys, which was published in *The Lancet* [11], we had Mrs. van Nuys undergo a brain MRI, an EEG and blood tests, of which only the MRI yielded a possible clue as to what was going on in her brain. As indicated by the MRI scan, her brain showed various discrete white-matter changes near the lentiform nucleus and in the semioval centre, which by themselves were not very specific in nature. Moreover, as they were not unusual for a person in her 50s, they might—under different circumstances—have been dismissed as insignificant. However, they were the only clues we had of what was going on in her brain, and we already knew that something was definitely going on there. Also, they looked as though they had been there for a very long time, possibly lifelong, which was also the case with Mrs. van Nuys’ perceptual symptoms.

The brain’s white matter consists of myelinated nerve-cell projections called axons, which connect the bodies of nerve cells that make up the brain’s grey matter. The function of grey-matter areas has been charted extensively (although more is yet to be charted), as well as the way they connect through tracts of white matter to other areas of grey matter. So,

whenever an MRI scan indicates the presence of a lesion in grey matter, we tend to have at least some idea of the function of those areas and thus whether the lesion might explain the patient's symptoms. However, regarding lesions in white-matter areas, things are much more complicated. For example, when we hear of a car accident somewhere between London and Birmingham, on the basis of that information alone, the emergency services will probably have no idea where they should be headed; there are simply too many roads between those cities to know for sure on which one the accident has happened. On MRI scans, areas of white matter show up as relatively blank areas—not even as road maps. This means that some guesswork is involved when dealing with white-matter lesions—although we do know that they should not be there in the first place.

Similarly, it was impossible for us to tell for sure whether there was any causal connection between the white-matter lesions in Mrs. van Nuys' brain and her perceptual symptoms. That said, we speculated that it was at least possible that they were due to a lack of oxygen during birth and that they had somehow disrupted the part of the visual network that has a function in the representation of faces. Since an important part of that network is the fusiform face area at the base of the brain, our first guess was that—if any lesions had been present—they would probably have been found there. Instead, finding changes in the white matter implied that we had no definite proof of what it was that had caused her symptoms. Despite the negative EEG findings, we were therefore inclined to attribute them to aberrant electrophysiological activity in the adjacent regions of the brain that have a function in representing colours and faces, located near one of the white-matter lesions in ventral occipito-temporal cortex .

What was not reported in the *Lancet* paper was that we had also made functional MRI scans of Mrs. van Nuys' brain. The reason why we hadn't mentioned that part of the investigation was that the ensuing images were of insufficient quality for publication; nevertheless, the test itself was revealing. Brain MRI scans yield images that represent the brain's anatomical structure. Functional brain MRI scans, however, yield images that indicate changes in oxygen consumption, which is taken as a sign of either increased or decreased activity of nerve cells in the brain. At that time, we had requested Mrs. van Nuys to take place inside the scanner and look at a series of images that we presented to her through a mirror system. Those images represented faces in various degrees of abstraction. We

showed her a smiley face, a rather vague charcoal drawing of a face, a realistic drawing of a face, a black-and-white photograph, a colour photograph, and a tree in which (albeit with some effort) a face could be discerned. Using a handheld air-pressure balloon, Mrs. van Nuys was able to indicate the beginning and ending of each episode during which she perceived any dragon faces.

What we found was that the balloon presses neatly coincided with episodes of altered activity in Mrs. van Nuys' visual cortical areas. From that, we concluded that her visual network was either more or less active during the perception of dragon faces. On the basis of fMRI images alone, one cannot tell one type of activity apart from the other. However, considering the fact that at those moments her brain was busy creating images of faces that were not actually there, we assumed that what we saw was hyperactivity not hypoactivity.

When the test was over, Mrs. van Nuys confessed that she had been very scared inside the scanner. We complimented her for her bravery, and for completing the test despite her grave discomfort. She then told us that seeing the tree with the hidden face had been the most difficult one for her. The smiley face had not evoked any dragon faces. The other images had, but the more realistic they were, the longer it had taken for them to appear. The tree face, instead, which left most room for her brain to create images of its own, had immediately evoked a veritable bombardment of dragon faces.

Afterwards, I discussed with Mrs. van Nuys what we had learned, and, more importantly, what could be done about the dragon faces. I explained to her that we were unable to provide any certain proof of anything but that her own hypothesis, that her visual symptoms had something to do with her being born with the caul, might well be correct. It was very well possible that the discrete white-matter lesions stemmed from a lack of oxygen in the brain during those minutes in which her head had been wrapped in birth membranes. If that was true, and if it was also true that those changes in white matter had indeed affected the part of the visual network involved in the representation of faces, this might explain why she had been seeing dragon faces all her life. Although that were a lot of 'ifs', since the faces always appeared after an interval of several symptom-free minutes, there had to be a functional aspect to her condition. Thus, irrespective of whether those white-matter lesions indicated that crucial parts of her visual network

had been damaged beyond repair, her brain had apparently found a way to work around that problem (at least temporarily); otherwise, it would have been impossible for her to ever see faces the way other people do, as she in fact did during the first minutes of contact with them.

As I already knew that antipsychotics and antidepressants had failed to help, I offered to prescribe something from an entirely different group of medicines, namely, the antiepileptic drug valproic acid . Mrs. van Nuys agreed and, when we spoke on the phone a week later, she said that, to her great relief, the dragon faces had bothered her less often. We agreed that she could increase the dose and, when we spoke again a few weeks later, she told me she had experienced several symptom-free days for the first time in her life. She was overjoyed—and so was I—because she had made it so clear to me how much she had been suffering from the dragon faces, and how much she had hoped to get rid of them.

Unfortunately, after this promising succession of symptom-free days, the faces returned, so I again advised her to increase the dose. Although this worked a third time, Mrs. van Nuys now reported that she heard loud bangs during the night, so loud indeed that they woke her up and really frightened her. I knew that this symptom was called ‘auditory sleep start ’ or, evocatively, ‘exploding head syndrome’ [12]. This, too, was a very rare symptom and its cause was unknown. As far as I knew, it had never been described before as a side effect of valproic acid; however, after advising Mrs. van Nuys to lower the dose, the noises gradually disappeared. This obviously suggested that they had had something to do with the valproic acid. However, whereas the noises were on the wane the dragon faces increased in intensity. Knowing that Oliver Sacks had had considerable experience with the antiepileptic gabapentin , and had successfully treated other functional brain disorders with this drug, I advised Mrs. van Nuys to taper off the valproic acid and try the gabapentin instead. Again, this had a positive effect on the intensity and frequency of the dragon faces, but unfortunately, the loud bangs increased simultaneously. I then offered to switch her to a totally different type of medication, the cholinesterase inhibitor , rivastigmine . This drug is registered for treating early stages of Alzheimer’s disease and had also been successfully used by our group in the treatment of musical hallucinations, another rare type of perceptual disorder [13]. Although Mrs. van Nuys did not suffer from Alzheimer’s disease or musical hallucinations, I found a few case reports in the literature

indicating that this type of medication could also be used in the case of visual hallucinations [14]. Moreover, since the visual system depends substantially on the neurotransmitter acetylcholine and rivastigmine tends to yield few side effects, I thought that it was at least worth a try. Mrs. van Nuys fully understood the experimental nature of this type of treatment and, realising that there were no precedents or evidence-based alternatives, she consented to it.

Although the night-time noises never completely disappeared, the rivastigmine did help Mrs. van Nuys to keep both the dragon faces and the noises down to a minimum. As we later wrote in our paper, this time she managed to hold on to her job for 3 years in a row. Two years after that, I learned that she had, nevertheless, had to quit her job due to irreconcilable differences of opinion with her manager; however, when Vivian Buijs interviewed her in 2016 for our follow-up study, Mrs. van Nuys was still on the rivastigmine and was still content that she perceived the dragon faces far less often than she had in the past. In the meantime, she had opened up a web store where she offered self-made jewellery and other artistic products, and also showcased various collections of photographs that she had made. It turned out that she had a good eye for composition—although I was somewhat surprised to find that she had a way with portraits, featuring people whom she knew as well as random people from the street. On second thoughts, however, I realised that it shouldn't have surprised me at all, that she—who had never been able to sustain a stable representation of human faces—had now seized the opportunity to capture them on film. In fact I could not think of anyone having a more dire need to study faces, with all their limitless variations and expressions, than Mrs. van Nuys.

These three case descriptions may serve as examples of how Alice in Wonderland is experienced in actual practice. In this book we will meet several other patients, with completely different stories to tell, even though they are all related in a diagnostic way. To appreciate why such diverging clinical pictures are subsumed under a single diagnostic label, we first need to outline the concept of Alice in Wonderland syndrome itself and see what it refers to—and why.

References

1. Nasar S (1998) *A beautiful mind*. Touchstone, New York

2. Howard R (2001) *A beautiful mind*. Universal Studios, Universal City
 3. Blom JD, Kooij JJS (2012) De ADD-psychose: behandeling met antipsychotica én methylfenidaat? *Tijdschr Psychiatr* 54:89–93
[PubMed]
 4. Yonebayashi H (2010) *The secret world of Arrietty*. Studio Ghibli, Tokyo
 5. ffytche DH, Howard RJ (1999) The perceptual consequences of visual loss: ‘positive’ pathologies of vision. *Brain* 122:1247–1260
[Crossref]
 6. Klee A, Willanger R (1966) Disturbances of visual perception in migraine. *Acta Neurol Scand* 42:400–414
[Crossref]
 7. Heron W, Doane BK, Scott TH (1956) Visual disturbances after prolonged perceptual isolation. *Can J Psychol* 10:13–18
[Crossref]
 8. Morehead DB (1997) Exacerbation of hallucinogen-persisting perception disorder with risperidone. *J Clin Psychopharmacol* 17:327–328
[Crossref]
 9. Sacks O, Blom JD (2012) Musical hallucinations. In: Blom JD, Sommer IEC (eds) *Hallucinations. Research and practice*. Springer, New York, pp 133–142
[Crossref]
 10. Blom JD, Looijestijn J, Goekoop R, Diederens KJM, Rijkaart A-M, Slotema CW, Sommer IEC (2011) Treatment of Alice in Wonderland syndrome and verbal auditory hallucinations using repetitive transcranial magnetic stimulation. A case report with fMRI findings. *Psychopathology* 44:337–344
[Crossref]
 11. Blom JD, Sommer IEC, Koops S, Sacks OW (2014) Prosopometamorphopsia and facial hallucinations. *Lancet* 384:1998
[Crossref]
 12. Pearce JM (1988) Exploding head syndrome. *Lancet* 332:270–271
[Crossref]
 13. Blom JD, Coebergh JAF, Lauw R, Sommer IEC (2015) Musical hallucinations treated with acetylcholinesterase inhibitors. *Front Psychiatry* 6:1–6
 14. Ukai S, Yamamoto M, Tanaka M, Takeda M (2004) Treatment of typical Charles Bonnet syndrome with donepezil. *Int Clin Psychopharmacol* 19:355–357
[Crossref]
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Footnotes

1 In this context, the term ‘halo ’ should not to be confused with the ring that we all tend to see on foggy nights around street lanterns or the moon. That type of halo is not a metamorphopsia but rather a physical illusion , which means that it is a type of misperception based on the laws of physics. Obviously, there is no actual ring out there. The light from the moon and the lanterns is dispersed by tiny droplets of water in the air and reflected in such a way that we perceive it as a ring-like structure surrounding them. The corona phenomenon , on the other hand, has nothing to do with the dispersion of light or any atmospheric circumstances.

2 Especially when epileptic activity confines itself to a localised, circumscribed brain area, even prolonged episodes may be too subtle in nature to register on an EEG . Moreover, Dimitri’s EEG had been made with scalp electrodes alone, which means that only the brain’s top layers had been explored and that epileptic activity, if present in deeper layers of the brain, might still have gone undetected.

3 I had worked with Sacks before on a book chapter on musical hallucinations [9].

3. The Making of a Syndrome

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

Authentic case descriptions such as those in the preceding pages are a convenient way to become acquainted with Alice in Wonderland syndrome and get a flavour of the ways in which it can affect people's lives. However, descriptions such as these can also be confusing, since the 'wheat' of the clinical picture needs to be separated from the personal, subjective, meaningful 'chaff' that makes them so authentic in the first place. Nevertheless, at least we now know what Alice in Wonderland syndrome is—right? If anyone asked you about it at this point, you would be able to tell them that it often involves animals; for example, people may see a rabbit (as Alice did), or a squirrel, or ants, or even dragons. They may become severely paranoid and agitated when you lower their medication, as happened to Dimitri, and things may go bump in the night, as happened to Mrs. van Nuys—and you certainly do not want to sleep in a crooked position in the car, or you might end up like Paul, seeing everything as slanted. Yet all these elements actually have nothing to do with Alice in Wonderland syndrome—although they certainly were peculiar phenomena, they were *not* perceptual distortions. Therefore, they needed to be ignored—even though they were an integral part of the stories that were told. They needed to be heard, understood, respected and reflected upon, but, in the final analysis, they needed to be side-lined for a while. That is what I meant by separating the wheat of the clinical picture from the chaff of these

personal, authentic accounts—and that is exactly what makes the making of a diagnosis such a delicate process.

Diagnosis stems from the Greek *diagignōskein*, which means ‘to discern’ or ‘to distinguish.’ Diagnosticians, in whatever field or specialty they work, need to come to terms with the fact that they interact with real, live human beings with unique personalities, abilities and characteristics, and with the fact that, somehow, they have to look beyond those unique human elements in order to discern the illness that lies hidden within their patients’ bodies, brains and minds. They have to forget about the human elements for a while, in order to get a glimpse of the disease process underneath, without becoming distracted by that which makes their patients human. Oliver Sacks was not only an excellent writer of medical stories, who knew how to populate his books with lovingly portrayed individuals—who all had something unique to share about the cards they had been dealt in life—he was also an expert at forgetting all that, at cutting through those stories and their all-too-human elements and tapping into the abstract configurations that we call ‘disease.’ That was what made him such an excellent diagnostician. Yet, what made him an excellent physician, above and beyond a mere diagnostician, was that once he was done tinkering with his diagnostic procedures, he was able to take a step back and see his patients as whole again, as the wonderful individuals that they were; in other words, not just ‘subjects’ suffering from this or that disorder—which, only moments ago, he had so expertly exposed—but human beings with abilities, talents, ways to cope with life, disease and death, and ways to make something out of it—no matter what. If diagnosis is indeed about overlooking the human elements, a true physician is also able to transcend that process after he or she is done—and allow it to be replaced by empathy.

If that is what characterises the diagnostic process, let us try to imagine what it takes to describe a *new* disease, to be a nosologist ¹ rather than a diagnostician. As a nosologist, one has to go through the same diagnostic cycle of meeting individuals whose bodies, brains or minds harbour some hidden illness; however, this time around, it is a hidden illness never previously described. Again, one needs to forget about the personal elements that make those people so special, unique and human in the first place; this time one needs to *imagine* (rather than recognise) what it is that they are suffering from, to *imagine* it in different individuals with different backgrounds and different stories, and then finally to *describe*, for the very

first time, the hidden pattern that connects these individuals in a medical sense.

Contrary to what you might perhaps expect, discovering a new disease is not a matter of uncovering some pre-existing process in the brain or looking through a microscope to find what was already there, like retrieving an Easter egg hidden by your parents in the garden. It is a matter of creating something new. That something new is a medical abstraction—and not just any medical abstraction but one that should lend itself for empirical testing and which, when it stands the test, yields novel possibilities of explaining—and hopefully also treating—that which people suffer from. In that sense, nosology is a creative process that draws on skills similar to those employed by painters of fine art, composers, couturiers, martial-arts masters, and industrial designers. It is through creativity—paired with solid medical expertise, of course—that diagnostic categories come into being, and those who design them are more like artists than prospectors. That certainly holds true for the person who first discerned the distinctive pattern of symptoms of Alice in Wonderland syndrome and gave the condition its name. That person was an extraordinarily keen physician named John Todd . If we wish to understand what Alice in Wonderland syndrome is, and why the concept was designed the way it is, we need to meet the man who introduced the medical world to it and hear what he had to say.

3.1 John Todd

John Todd (1914–1987) was a British consultant psychiatrist who spent most of his working life at High Royds Hospital , near Menston, West Yorkshire. Originally known as the West Riding Pauper Lunatic Asylum , the hospital had been founded in 1818, at the beginning of what we call the era of classical psychiatry (Fig. 3.1). After having served its purpose as a mental healthcare institution for almost two centuries, it was closed down in 2003. Some of its quarters have since been converted for residential use, but in Todd’s days (from the 1950s through the 1980s), the large complex of gothic-style stone buildings and its surrounding Yorkshire estate were alive with business—housing a more or less self-sustaining community of physicians, nurses, librarians, gardeners, cooks, butchers, bakers, upholsters, cobblers and men and women of many other trades, and where patients (if they were up to it) were given specific tasks to contribute to the

activities. There were even facilities for leisure and entertainment, including a small film theatre and, every Friday night, there were festive events in the great ballroom, where male and female patients were allowed to meet each other and dance the night away—an event only topped by the Christmas asylum ball, which attracted some 800 patients every year [1, 2].

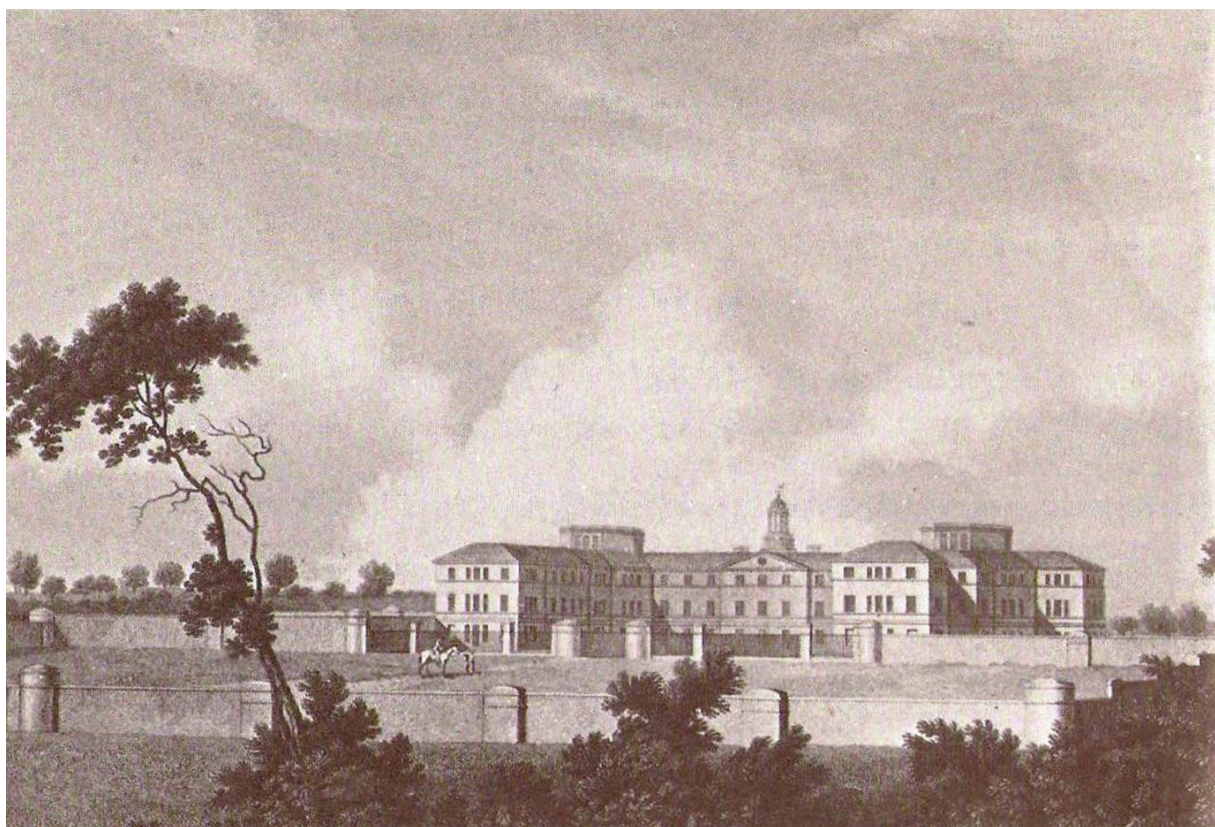


Fig. 3.1 The West Riding Pauper Lunatic Asylum, Landseer engraving of 1818. This is where John Todd used to work as a consultant psychiatrist from the 1950s through the 1980s

However, that was as cosy as it got at the Menston asylum. Above all, it was a place where psychiatric patients had been tucked away, isolated from society. They were suffering from God-knows-what, hearing voices, having visions, writhing in agony from poorly understood assaults on their nervous system, bouncing from the screaming highs of manic episodes into the bottomless pits of depression and trembling from fear or from the withdrawal of chloral hydrate that was generally used to treat it. Moreover, they were always at risk of being assaulted by some deluded soul who might get to them before the staff could intervene and pull them apart, or from ending up in solitary confinement themselves because their behaviour

was not understood or simply not tolerated, whether it had been of an aggressive nature or not. In many parts of the asylum, the doors were locked; to pass through them, one needed permission. One was always at the mercy of staff, and one was always under observation, even during the weekly mixed-dancing outings.

By the time Todd arrived there, chlorpromazine , the first medicine marketed as an ‘antipsychotic ’, had also arrived. It was developed in France, and although it did not cure psychosis , it proved so effective in managing psychotic symptoms that it was quickly implemented internationally. Since even patients with long-lasting symptoms could benefit from it, eventually, this new therapeutic tool allowed for a veritable exodus out of the old institutions, the majority of patients having become sufficiently stable to be returned to society. Needless to say, those who stayed behind were as ill and disabled as any of these patients had ever been and, moreover, largely medication-resistant. Thus, the patient population that awaited Todd when he came to High Royds Hospital consisted of the most severely ill individuals imaginable, often difficult to manage and extremely difficult to treat. Inevitably, among them were also a number of people suffering from disorders that no one had ever heard of, as well as from disorders that had not yet been described.

Todd was originally from London, where he had been educated at the City of London School and King’s College Hospital. As a young man, he must have been the epitome of a sound mind in a healthy body, as he played chess for the university team, won the Tanner prize for obstetrics and gynaecology and also won the London University colours for boxing. Following in his father’s footsteps, he qualified as an MD in 1938. Two years later, he entered the British Royal Army Medical Corps and, in 1944, took part in the Normandy landings , one of the grimmest military endeavours of World War II . After the war, he found himself back in England, having to choose a medical specialty of his liking. The prize that he had won for obstetrics and gynaecology must have been a strong incentive to steer his career in that direction, but apparently, it was psychiatry that fascinated him the most. Consequently, in 1946, he started his psychiatric career at Park Prewett Hospital , Hampshire, from where he went on to Littlemore Hospital , Oxford. During a stay at the nearby Radcliffe Lunatic Asylum (now Warneford Hospital), he met Caroline Harvey (1926–2006), a medical secretary with whom he would marry and

go on to have a son and two daughters. In 1955, Todd accepted a senior position at High Royds Hospital (then still called the West Riding Pauper Lunatic Asylum), whereupon he and his young family took residence in a villa on the hospital grounds. It was there, in the bubble of that secluded country community, amongst this group of tragically ill and isolated patients, that he stayed for the remainder of his career and became known for his personal service to them, striving for a kind of 'personalised medicine' before the term had even been invented. His specialty was diagnosing and treating those whose disorders were either too particular or too complex to fit in with any of the conventional diagnostic categories. His devotion soon earned him a reputation among his colleagues, who started referring to their own complex patients as 'a Todd', and were all too glad when he was willing to see them for a consultation.

Todd's taste for the exotic must have caught on at an early stage of his career because, while still in the residency training programme at Littlemore, he had written the paper that would put Alice in Wonderland syndrome on the map. That paper was published on November 1, 1955, in the *Canadian Medical Association Journal* [3], in which his father had published before him. It was one of John Todd's many outpourings on rare and unusual psychiatric disorders, which one could encounter each and every day at the Menston Asylum, provided that one had an eye for them. Although most of these disorders² had been described before, Alice in Wonderland syndrome was Todd's own creation. As he wrote in his 1955 paper, he had come up with the term '...to draw attention to a singular group of symptoms intimately associated with migraine and epilepsy, although not confined to these disorders' [3]. To conceptualise this new syndrome, he drew inspiration from Lewis Carroll's descriptions of Alice's peculiar perceptual changes. As he wrote,

It will be remembered that Alice, in her dreams, sometimes becomes remarkably tall or remarkably short. However, she was sometimes aware of changes of an altogether more subtle nature. Thus, there were occasions when she was conscious of some intangible change in herself and her environment. There were also times when she addressed herself as though she were two people, and others when she puzzled over her own identity. In technical

terms, she had feelings of hyperschematia , hyposchematia , derealization , depersonalization , and somatopsychic duality . [3]

Before I go on to explain these terms and illustrate how the various symptoms are experienced by actual patients, let us see what else Todd considered characteristic of his novel disease category. As he went on,

There are good reasons for including certain other symptoms within the general purview of the syndrome; they include illusory changes in the size, distance, or position of stationary objects in the subject's visual field; illusory feelings of levitation ; and illusory alterations in the sense of the passage of time . [3]

Thus, according to Todd, Alice in Wonderland syndrome comprised the following groups of symptoms:

- Distortions of bodily sensation
 - Hyperschematia and hyposchematia
 - Somatopsychic duality
 - Visual distortions
 - Time distortions
 - Illusory feelings of levitation
 - Derealisation
 - Depersonalisation

Incidentally, he was not the first to describe these individual symptoms. Others had published on them before, and even parallels with *Alice's Adventures in Wonderland* had been drawn before [4, 5]. What Todd deserves credit for is that he grouped them together, recognised their interrelatedness as perceptual *distortions* rather than hallucinations or illusions and endowed that group of symptoms with a name.³ And what a name it was: by calling it 'Alice in Wonderland syndrome', Todd secured himself of a memorable tag that would prevent it from disappearing into oblivion. He thereby brought a new diagnostic category into being, a medical subject worthy of studying in its own right and a reality in the lives of people who had until then been suffering from symptoms that proved elusive to themselves *and* to the majority of health professionals. Most importantly, perhaps, by using the term Alice in Wonderland syndrome, he

found a way to immediately conjure up the types of symptoms he considered characteristic, since we all know what kind of weirdness Alice had to put up with in Carroll's story . Contacts with his patients had convinced Todd that these phenomena belonged together, like pieces of a puzzle that laid scattered throughout the *Alice* book—and that, together, they formed a meaningful whole.

Such a meaningful whole is what we call a syndrome . In medicine, this term is used to convey the suspicion that certain signs and symptoms belong together because they have a tendency to occur in combination with each other or because some other relation is assumed. Down's syndrome , for example, the well-known triad of intellectual disability, physical growth delay and characteristic facial features, was first described in 1866 by the British physician John Langdon Down (1828–1896) because it had become apparent to him that these symptoms often occurred together. Having established that, Down then inferred that some common hereditary factor must be at play. Since genes, chromosomes and DNA were all unheard of at the time, it took almost a century before a group of physicians, headed by the French paediatrician Jérôme Lejeune (1926–1994) [7], was able to confirm that Down's syndrome is indeed best explained as a genetic disorder, caused by the presence of a third copy (in whole or in part) of chromosome 21.⁴

Whether some similar common cause may underlie all cases of Alice in Wonderland syndrome is unknown. As we shall see in Chap. 5, numerous medical conditions may trigger it, including epilepsy , migraine and substance abuse . However, the majority of patients suffering from epilepsy, migraine or substance abuse do not go on to develop Alice in Wonderland syndrome. Thus, perhaps, a special vulnerability is indeed needed to promote Alice in Wonderland syndrome, even in the presence of such disorders. Following that line of thought, it is not unthinkable that Alice in Wonderland syndrome may also prove to have a genetic basis, in the sense that the vulnerability to develop perceptual distortions may be hard-wired into our genes. Nevertheless, even apart from that possibility, it is of crucial importance to be able to distinguish the symptoms of Alice in Wonderland syndrome from other positive disorders of perception. After all, if doctors systematically mistake them for something else, how are they to recognise them in clinical practice and treat their patients properly and appropriately?

Overall, the virtue of Todd's concept is threefold. First, it draws attention to the group of perceptual *distortions*, which have been neglected for so long by so many health professionals. Second, it points the way to numerous medical conditions that may underlie them. Third, by using the word syndrome, it silently implies the possibility of a certain vulnerability (genetic or otherwise) shared by those who suffer from such symptoms. We will return to these neurobiological implications in Chap. 5; however, let us first examine in more detail what kind of symptoms Todd referred to and how they are experienced in clinical practice.

3.2 Symptoms of Alice in Wonderland Syndrome

The symptoms characteristic of Alice in Wonderland syndrome belong to what we call the 'third group of positive disorders of perception'. They are neither hallucinations nor illusions, which make up the first two groups. Thus, they are different from hearing voices when there is no one around, from seeing animals that are not really there, from smelling odours that others do not smell and from taking a moving curtain for an intruder—to mention just a few of the symptoms with which they might easily be confused. What sets them apart from hallucinations and illusions is that they are *distortions* of regular sense perceptions. What people with Alice in Wonderland syndrome perceive is something real, something out there in the physical world, which, however, presents itself in a twisted, warped, contorted manner.

3.2.1 Somesthetic Distortions

The first distortions mentioned in Todd's 1955 paper are somesthetic in nature, which means that they affect the way we experience our body [3]. When Todd speaks of Alice becoming remarkably tall or remarkably short, he refers to that which neurologists call macro- and microsomatognosia, a pair of symptoms we encountered before. As you will remember, Mr. Salvatore had been suffering from *partial* microsomatognosia for over 35 years, feeling his hands being reduced to the size of tiny stumps whenever he put them in his pockets, while I myself had once experienced the opposite sensation of partial *macrosomatognosia* when I was down with a fever, feeling my hands growing to the size of balloons. Looking back, I am glad that I had had a chance to experience that because, years later,

when I had a daughter who was then 10 years of age, she, too, was down with a fever and she, too, experienced partial macrosomatognosia.

In Esther's case, the fever had not been due to food poisoning but to a bout of flu . So, Renate and I kept her from school that day, put her on the couch in the living room, tucked her in under a warm blanket, brought her the pillow from her own bed, provided her with enough comics, cartoons, water and liquorice (we are Dutch) to entertain a small children's party and took turns staying home from work to be with her. Thus settled in, Esther no longer seemed to mind the fever and had simply lain there, quietly enjoying her unexpected day off, with only blushes on her cheeks to betray the nature of her condition. So, being there with her and everything being cosy, I was surprised to notice at some moment in the afternoon that she suddenly had tears in her eyes. I went up to her, sat down next to her on the couch, and asked, 'What is it, sweetie, are you in pain?' Upon which she whispered, 'No, daddy, but—.' Then she paused, looking at me with tear-stained eyes, to continue, 'My hands feel so large, and my legs feel as if they are so very, very long'.

That, of course, was where my own experience came in handy because I was able to tell her that I had once had the same feeling (except for the legs) and that it was a fairly normal thing to have during a fever and that it would probably soon be over—even though I knew that it does indeed feel really, really weird. That afternoon she continued reading, and sleeping, and watching films and, in the evening, when the fever had subsided, she confirmed that her hands and legs had returned to feeling normal again.

I was glad that I had been able to reassure her and tell her that it is not such a very strange thing to experience. At least, that is what I hear from people in my vicinity, and from those who attend my lectures, or send me feedback on my papers. Some time ago, for example, I received an email message from a man who had been reading some of my work; he told me that years ago he had been hospitalised because of an unnamed infectious disease and, lying in bed in a six-person hospital room, had suddenly had the feeling that his hands were growing so large that they seemed to fill up the entire room. It had not been a painful experience, he added, just a very peculiar one; moreover, he had soon discovered that all he could do was sit the feeling out until it gradually disappeared—parallel with the improvement of his condition.

3.2.2 Hyperschematia and Hyposchematia

In his paper, Todd subsequently speaks of hyperschematia and hyposchematia . With those terms, he refers to changes in the way we experience the space occupied by our bodies. *Hyperschematia* is an exaggeration of the space occupied by one's body. Although neurologists know this symptom chiefly as a left-sided phenomenon (due to a stroke or other lesion to the right hemisphere of the brain), it may affect either side of the body, and even the body as a whole. Think of pushing your hand into a fresh pack of snow and having to estimate the size of the print that it will leave. If you suffer from *hyperschematia* , you will estimate it to be larger, and in the case of *hyposchematia* , you will estimate it to be smaller than the print will actually be. A clinical example of hyperschematia comes from Todd's paper, in which he describes a 39-year-old woman who had worried since childhood about recurring changes in the way she experienced her body:

She complained of recurrent attacks during which she feels that her body is growing larger and larger until it seems to occupy the whole room. 'I feel,' she said, 'that I have got so big that if I put out my hand I could touch the far wall' [3].

This woman suffered from total-body macrosomatognosia , feeling her body 'growing larger and larger', while simultaneously, as a complementary symptom, she suffered from hyperschematia, as exemplified by the sensation that she could 'touch the far wall' by simply putting out her hand.

I myself had a somewhat similar experience when I was a young doctor and had just obtained a new pair of glasses. Obviously, I did not develop Alice in Wonderland syndrome all of a sudden, but the new glasses nonetheless played a remarkable trick with my mind. Although I had been wearing glasses since my freshman year at university, this time I had been told that my eyes were astigmatic; hence, the lenses had been polished in a cylindrical fashion, yielding a gradient of increasing power in opposing directions. While testing them at the optician's, I had peered at distant objects (such as advertisements on the counter, and people and buildings in the street) only to find that I saw things a little smaller than I was used to but with a greatly enhanced visual acuity. Thus, for the first time in years, I

was able to clearly read the names on shop windows across the street, and I even remember how much I admired the level of detail that I could now discern in the store's carpet piles. So I thanked the optician for his excellent work, paid the bill, put on the glasses, walked out the door, stepped on my bicycle and, to my astonishment, found that I appeared to have stepped onto the old tricycle that I had when I was 4 years old, sitting really close to the ground, with legs that suddenly appeared to be ridiculously short. When I looked around me at other cyclists, their heads were at the same level as mine, but they all looked shorter than usual, as if I were looking at a film with a messed-up aspect ratio. Riding home, I could not shake the sensation of paddling forth on a child's bicycle. At home I tried walking in the backyard—and it was like walking on the shallow side of a swimming pool. Looking down at my legs, they seemed oddly short and, by comparison, my arms seemed oddly long, with my hands almost touching the ground.

Because I had never worn cylindrical lenses before, my brain had to adjust to this new type of visual input. As a result, I temporarily suffered from hyposchematia, estimating the distance between my waist and the ground as being much smaller than it actually was. When I took the glasses off, I saw everything as blurry as I used to—but at least experienced myself and my surroundings in familiar proportions. However, for the next 3 days, whenever I put the glasses back on, it was as if I were wading through shallow water, with oddly shortened legs and arms extended like those of a monkey. After those 3 days, my brain apparently got the message and, from then onwards, everything was back to normal.

It was a weird experience. Yet I considered it rewarding since it made me realise—in a harmless way—how disturbing hyposchematia can be and allowed me to get an idea of how frightening the experience could have been, had its cause not been so obvious and had things not returned to normal so easily.

3.2.3 Derealisation and Depersonalisation

In his paper, when Todd subsequently speaks of derealisation and depersonalisation, we are back to the symptoms Mr. Salvatore had suffered from so badly. As we saw, derealisation is a distorted sense of reality—although it does not need to be so creepy as it was to him. When you walk out of a movie theatre into broad daylight and suddenly feel overwhelmed by the crowds and the noise, and the light that seems to bounce off from just

about everything, you also experience derealisation. You will probably recognise the feeling because everyone experiences it from time to time, be it while undergoing an abrupt change of scenery, while working under stress, or following some traumatic event. My experience with the large hands, and the other one with the mini-bicycle and the monkey arms, gave me a foretaste of what more severe types of derealisation must be like.

Depersonalisation, on the other hand, is the sensation of not being there, of experiencing oneself in an oddly detached way. Patients often tell me that they experience themselves as if they see the world from behind a thick pane of glass. Others tell me that they experience the world as if from a position near the back of their head, as if they were looking out into the world while they themselves were seated in a tiny chair, deep inside the recesses of their mind, observing all that is going on as if projected on a giant screen. Sometimes, they may even need to check their own presence, as described so aptly by Todd, who wrote the following about one of his patients:

Without warning, she would be overwhelmed by feelings of unreality of such intensity that she was compelled to look in a mirror to confirm her presence in the room. [3]

From that, insecurities may arise about one's personal identity. As Todd tells us about yet another patient:

There were occasions when she was conscious of some intangible change in herself and her environment. There were also times when she addressed herself as if she were two people, and others when she puzzled over her own identity [3].

3.2.4 Somatopsychic Duality

Here we arrive at somatopsychic duality, which appears to border on the classical concept of multiple personality disorder; however, this is an altogether different symptom. It is definitely not a Dr. Jekyll and Mr. Hyde kind of situation, but rather the experience that one does not reside in a single place. In his paper, Todd described a 24-year-old woman who had had the transient sensation of being 'split' which, in her case, was accompanied by the sensation of having a second head. Another patient of

his, a girl aged 17 years, sensed the presence of an invisible alter ego, described by her as ‘an invisible double stationed a yard away on my left. This shadowy double seems to contain my mind’ [3].

3.2.5 Visual Distortions

Further symptoms mentioned in Todd’s paper include *illusory changes in the size, distance or position of stationary objects* in the person’s visual field; these are what we call metamorphopsias , or visual distortions. As seen in Appendix A, Table A.2, many different types have been described in the medical literature. We will examine them in more detail in Chap. 5, but we already encountered Ms. Artemis’ gyropsia (seeing stationary objects as if rotating), Mrs. van Nuys’ prosopometamorphopsia (seeing faces change), Paul’s plagiopsia (seeing things as slanted) and Dimitri’s corona phenomenon (seeing an extra line or contour around objects). Included in this category are also now familiar phenomena such as micropsia and macropsia , as well as kinetopsia (seeing movement that is not there) and akinetopsia (the inability to see movement).

3.2.6 Illusory Feelings of Levitation and Time Distortions

Todd then goes on to mention the *illusory feeling of levitation* , which is the strange sensation that one is floating in the air and, finally, *illusory alterations in the sense of the passage of time*. These are perhaps best illustrated by revisiting *Alice’s Adventures in Wonderland*, where they feature rather prominently.

3.3 Symptoms in the *Alice Book*

With the eponym, Alice in Wonderland syndrome, Todd explicitly acknowledged his indebtedness to the book written by Charles Dodgson . As we have seen, Todd’s paper listed several examples of what had inspired him, but, the other way around, we may ask ourselves which of those symptoms had actually been described by Dodgson in his famous book, and how.

In the book, we find Alice musing quite early on that things are getting ‘curiouser and curiouser’, while we, the readers, already know that this is only the beginning and that she will soon find herself immersed in a world with unanticipated levels of weirdness. In the pages to follow, she will meet

a Caterpillar puffing a hookah, a sobbing Mock Turtle , a bunch of live flamingos that are used for mallets while live hedgehogs serve as croquet balls, a pack of cards posing as gardeners busying themselves with painting the roses, a Kafkaesque trial at the court of the King and Queen of Hearts , a collection of little bottles and cakes that appear out of the blue, a Mad Hatter who rambles on about time , a Dormouse that talks in its sleep, a March Hare that gets raving mad each March and, in fact, so much more that it is hard to keep track of all the nonsense and not join in wholeheartedly with Alice's weary sigh. Then there are the animals, the numerous animals, all of whom are able to speak—except for the overgrown puppy (which actually only yelps and tumbles head over heels in its hurry to get hold of a stick), displaying such stereotyped puppy behaviour that in the context of the bonkers story, it stands out as just another bonkers element (Fig. [3.2](#)).



Fig. 3.2 Alice and the Puppy, illustration by Sir John Tenniel (1890)

It all starts out with the White Rabbit , of course, who, in perfect symmetry with the squirrel that heralded Ms. Artemis' brush with Alice in Wonderland syndrome, provides a fittingly absurd introduction to all the other absurd things to come (Fig. 3.3). It may, therefore, be tempting to think that seeing animals, or hearing them speak, is an integral part of Alice in Wonderland syndrome; as we saw, however, that is not the case. The syndrome is about perceptual *distortions* not hallucinations , and therefore, the White Rabbit does not count as an example.



Fig. 3.3 The White Rabbit, illustration by Sir John Tenniel (1890)

3.3.1 Illusory Feelings of Levitation

One of the first scenes in the book is about Alice falling down the rabbit hole, very, very slowly—so slowly, in fact, that she has plenty of time to look about her and take a jar labelled ‘ORANGE MARMALADE’ from a shelf and, finding it empty, stow it away in a cupboard a little further down.

This corresponds with the *feeling of levitation* that was described by several of Todd's patients, a distortion of our sense of movement and of our position in space. This symptom is indeed considered characteristic of Alice in Wonderland syndrome. So that is number one.

3.3.2 Somesthetic Distortions

The next symptom described in the book is Alice experiencing herself to be altering in size. This happens on 12 different occasions, starting with the scene in the long, low hall, where she finds a glass table with a tiny golden key on it that fits in the lock of the little door that leads into the 'loveliest garden she ever saw'. Being unable to fit herself through the door, she returns to the glass table and then finds a small bottle on it with a paper label tied around its neck, saying, 'DRINK ME'. After verifying that the bottle is not marked 'poison', she tastes its contents and then finishes it off. The effect, as we all know, is that she grows smaller, shutting up like a telescope, until she is no more than 10 inches high and can now easily pass through the tiny door leading into the garden—provided that she is still in possession of the golden key which, alas, she has left on top of the glass table, which is now out of her reach. After she has started crying, she pulls herself together and finds a glass box underneath the table containing a small cake, marked with the words 'EAT ME' in currants. Upon eating it, she finds herself opening out 'like the largest telescope'. If the first instance of change had been a case of *total-body microsomatognosia*, this one is its logical counterpart, called *total-body macrosomatognosia* [8]. A third instance follows soon thereafter, when Alice is fanning herself with the fan dropped by the White Rabbit and, as a consequence, finds herself growing smaller again. When later on she has entered the White Rabbit's house, she drinks from another little bottle and then grows so large that she gets stuck in the room, with her head pressed against the ceiling, thereby feeling forced to put one arm out of the window and one of her feet right up the chimney. Being threatened by the tiny creatures that besiege the house, she then desperately wants to grow small again. To her aid come a shower of little pebbles that rattle in through the window, some of them hitting her face, and turning into little cakes. Swallowing one of the cakes sets off the fifth transformation, which involves shrinking again. She then flees the house, meets the giant puppy and, after some more wandering about, runs

into the Caterpillar , who quietly sits smoking his hookah on top of a large mushroom (Fig. 3.4).

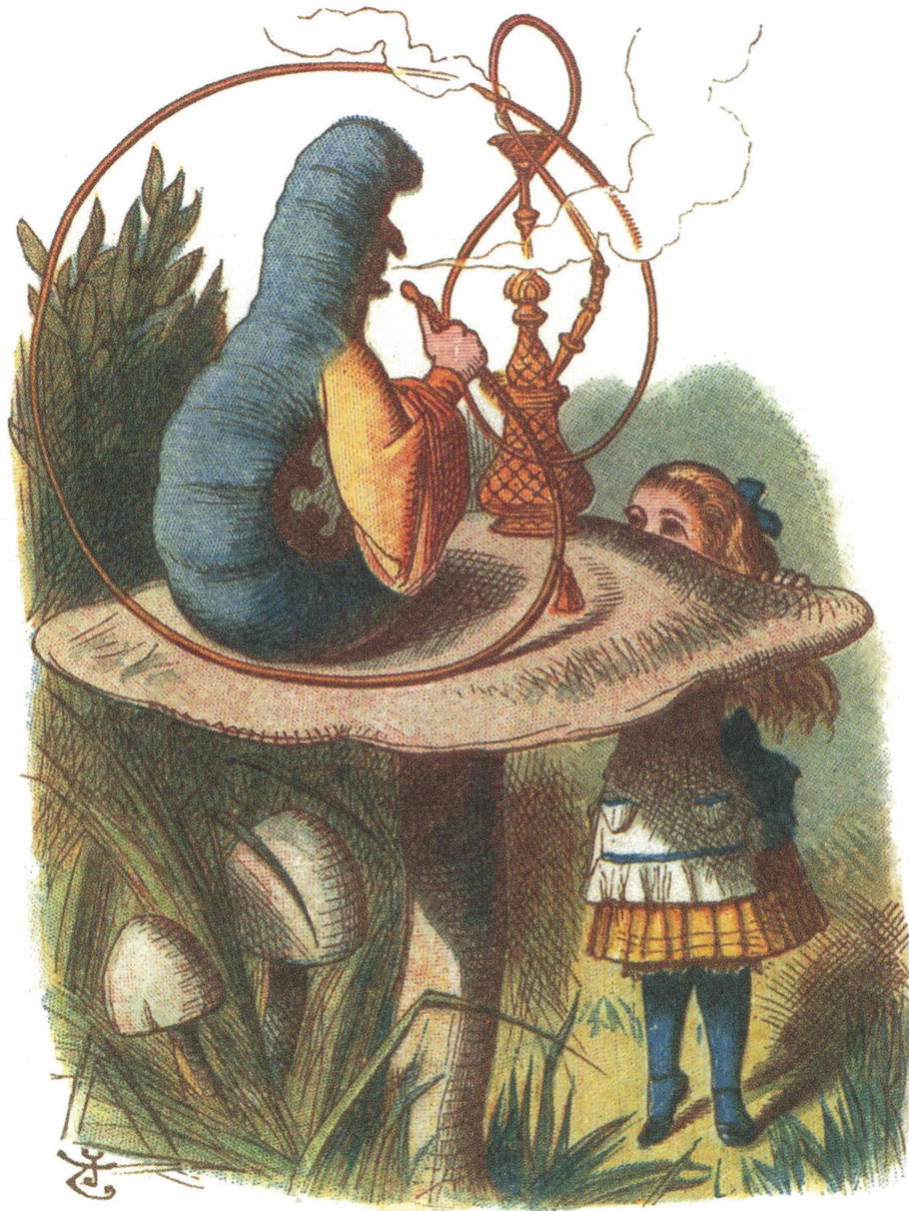


Fig. 3.4 Alice Looking at the Caterpillar, illustration by Sir John Tenniel (1890)

Upon finishing his interrogation, he leaves the scene and, after having called out to her that ‘One side will make you grow taller, and the other side will make you grow shorter,’ Alice follows this curious piece of advice and stretches her arms around the mushroom, thus managing to break off bits from opposite sides with both hands. Even though it doesn’t make any

sense to speak of two sides of a circle, by subsequently eating from the piece of the right-hand side, she shrinks even further. This sixth instance of change is even more peculiar than the previous ones, as Alice now shrinks so much and so fast that her chin strikes her foot, which indicates that she has not only grown smaller, but that the proportions of her body have also gone awry. Therefore, this transformation involves a combination of partial *and* whole-body microsomatognosia, making Alice's trunk and legs shrink even faster than her head and feet. Luckily for her, she is still holding bits of the mushroom from both sides in her hands—which indicates that her hands have not grown too small—and, after eating a bit from the left-hand side, the seventh change involves growing large again: so large, however, and again so much out of proportion, that her neck rises like a stalk above the trees from where she is unable to locate her shoulders. To her relief, this combination of partial and total-body macrosomatognosia is as weird as it gets for her body image. At that point she seems to silently declare herself to be in charge of the situation and, by carefully nibbling pieces from the left and right-hand side of the mushroom, she manages to bring about the eighth transformation, which involves restoring her usual height. Having thus regained her proper size, as well as her self-confidence, she then deliberately makes herself smaller (the ninth transformation) by eating bits of mushroom from the right-hand side to prevent herself from frightening the creatures that she is about to meet at a little house in the woods (which turns out to belong to the Duchess). After having stayed with the Duchess and her violently insane staff (while remaining at a height of 9 inches), she then deliberately makes herself somewhat larger again, 2 feet high, by eating bits of mushroom from the left-hand side, before walking into the Mad Tea Party. This is the tenth occasion on which her body size is altered. When, still later, she passes through a door in a tree to finally return to the long hall where she was at the beginning of the story, she knows exactly what is needed to unlock the little door and at last enter the beautiful garden that she had been longing for so much:

Once more she found herself in the long hall, and close to the little glass table. 'Now, I'll manage better this time,' she said to herself, and began by taking the little golden key, and unlocking the door that led into the garden. Then she set to work nibbling at the mushroom (she had kept a piece of it in her pocket) till she was

about a foot high; then she walked down the little passage: and then - she found herself at last in the beautiful garden, among the bright flower-beds and the cool fountains [9].

This is the 11th transformation, brought about so expertly by Alice that we are inclined to believe that she is now in total control of the process—at least until near the end of the book, when the 12th and final transformation kicks in during the Court scene and Alice spontaneously starts to grow, and grow, and grow, until she becomes a towering figure among the little creatures (who start scrambling for cover) and goes on to find herself waking up in her sister's lap (for, as Dodgson then reveals, it had all been a curious dream, you see).

As we saw, sensations such as these also happen to persons in real life, in the sense that they may experience their body *as if* it were changing in size, in part or in whole. Over the years, I have met several patients who described this phenomenon, mostly during brief attacks. We already met Mr. Salvatore. Another example is Ms. Rembrandt, a woman who had contacted me after she had seen footage of my lecture for the Dutch television programme *Universiteit van Nederland* and explained to me through email that she had been diagnosed with bipolar disorder, migraine and borderline personality disorder and had now become convinced that, on top of all that, she also suffered from Alice in Wonderland syndrome. I must admit that, at first, I had been somewhat sceptical because her email had been rather lengthy and had given me the impression that she was probably too confused to sort out what was what for herself. That impression was heightened over the next few months by the fact that she had requested a consultation with me but subsequently failed to provide me with the necessary documentation about her medical history, which was indispensable if I were to do my job properly. I don't exaggerate when I say that for almost half a year, we emailed to and fro, and each time that I requested additional information about prior hospital admissions, consultations with neurologists and so on, I would receive mere snippets of information, such as a single page from a follow-up file, an incomplete letter, or a list of medication that stopped halfway through. Sometimes I would even get emails enquiring whether I had received something, whereas my mailbox indicated that nothing of the sort had come through. Even contacting her family physician turned out to be of no avail, as most

of the information that he forwarded to me was already in my possession—and still incomplete. When at last not much else appeared to be trickling my way and I had given up hope that any further delay would do anybody any good, I sent Ms. Rembrandt an invitation with the intention of hearing her out and then referring her back to her own psychiatrist, as I expected there would be very little that I would be able to do for her.

How prejudiced one can be...

To my surprise, I met with an intelligent, well-composed woman in her 40s, who told a tragic story that started in her early childhood with ‘spells’ during which she would ‘roll over like a ragdoll’, as her mother had told her afterwards, and would end in her losing consciousness. As her mother had two sons with epilepsy, she took her daughter to a child neurologist, who confirmed the suspicion of epilepsy and prescribed antiepileptic medication. This apparently solved the problem, as she no longer lost consciousness. For how long she remained on this medication she could not remember, but after it had been tapered off (around her 5th or 6th year), she started having different spells, during which she perceived the world as being distorted, and her body as a tiny, worm-like object dangling under a hugely bloated head, furrowed with deep folds and creases, and covered with bumps. Meanwhile, her hands would grow very long and turn into a kind of witches’ claws, with long nails and all. During these attacks, she was unable to speak, often fell down and then kept lying on the floor or wherever she had landed, curled up like a foetus. At the age of 21 she had become so desperate that she made her first suicide attempt. She survived, though, and told no-one; after this, she struggled on to make the best of her life for 2 more years before she was admitted for the first time to a psychiatric hospital because of depression, weight loss, anxiety attacks and what was perceived to be hyperventilation. All the while she kept having her ‘spells’; however, now they were interpreted as anxiety attacks and then, later, as ‘pseudohallucinations’, an awkward term that usually says more about the level of doubt on part of the person using it than about the alleged ‘pseudo’ nature of the symptoms. Several admissions followed, whether or not after suicide attempts, which she kept carrying out from time to time out of utter despair. Throughout the years she was consecutively diagnosed with depressive disorder, anxiety disorder, eating disorder, borderline personality disorder and bipolar disorder and treated with all types of medication regularly prescribed for those conditions. Only for the past 7

years had she succeeded in living a more or less stable life on lithium (a mood stabiliser), sumatriptan (a medicine against migraine) and a long list of adjuvant medications.

When I finally saw Ms. Rembrandt at the outpatient clinic, she was working as an experience expert in psychiatry and, having gone through a divorce, had a shared parenthood over her daughter with her now ex-husband. Meanwhile, the spells had continued to plague her several times a week, from childhood onwards, up until that very day. During those spells, she not only felt her body transform but also perceived movement in the walls (kinetopsia), saw the room as bigger than it was (macropsia), saw objects taking on the colour and pattern of neighbouring objects (illusory visual spread), saw fluorescent yellow lines around objects (corona phenomenon), saw colours more brightly than they actually were (chromatopsia) and saw stationary objects continuously moving in her direction (macroproxiopia). During those attacks, when she had not ended up on a floor somewhere and was able to look in the mirror , she saw her face as hugely distorted, with a tiny mouth and a grotesquely bloated forehead that was covered with bumps, separated from each other by deep furrows. When she looked at other people, though, she saw no apparent changes in their faces.

As usual, I sat down with her behind the computer to have a look at a PowerPoint file with representations of all the different symptoms of Alice in Wonderland syndrome, and she was very pertinent about which symptoms she recognised and which not. My psychiatric evaluation revealed nothing out of the ordinary, except for the reported metamorphopsias and bodily symptoms of Alice in Wonderland syndrome, followed by what I took to be absences , or else severe instances of derealisation or dissociation . Her mood was perfectly stable, neither manic nor depressed, and she displayed no signs whatsoever of the psychodynamics typical of a borderline personality disorder . In short, here was a woman in her 40s who had battled with Alice in Wonderland syndrome all her life, who had weathered the storms of childhood epilepsy , had subsequently suffered from incomprehensible ‘spells’, mood swings and migraine attacks, had endured numerous admissions to psychiatric hospitals (with all the traumatising events generally associated with such interventions), had survived several suicide attempts, had lost the job that she had had as a young woman (as a caretaker for dementing elderly), had

seen her marriage get shipwrecked, was allowed to see her daughter for only half of the time and now sat in front of me without a single complaint about the course that her life had taken, just glad to find out that she had been right when she had diagnosed herself with Alice in Wonderland syndrome. I, for my part, only had the deepest respect for the way she had dealt with all that.

So there we sat, letting the consequences of our joint conclusion sink in. The obvious question that hung in the air was—what could be done about this bizarre situation. I explained to Ms. Rembrandt that the epilepsy and the migraine were both likely causes of her Alice in Wonderland syndrome. I then referred her to a neurologist to ask his opinion and have auxiliary investigations carried out. She agreed with that and, 6 weeks later, the results came in. The MRI was clean. The EEG showed some aberrant activity, although not the spikes or sharp-wave complexes characteristic of epilepsy. As the EEG had not been made during an attack, I considered it to be inconclusive and proposed nevertheless to prescribe antiepileptics, as this had apparently been successful during her childhood years, and the attacks followed a pattern that was compatible with epilepsy.

After 2 weeks on a low dose of valproic acid, I received an email saying that, for the first time since her 5th or 6th year, Ms. Rembrandt had been free of any attacks. She could hardly believe this to be the case—she slept better, woke up well-rested, had a clear head, and no longer suffered from any of the symptoms of Alice in Wonderland syndrome that had been wreaking havoc in her life for so long. It was also a bit eerie, she texted, having been accustomed to this life that she had always been living, and now experiencing the world with so much more tranquillity and peace.

When she came to visit me once more in The Hague, she thanked me for all that I had done (which had not really been that much, considering that she had essentially established her own diagnosis) and told me that I could certainly use her story for research or educational purposes if I so wished. I thanked her for that and said that I would gladly do so. Then, she gave me a painting in which she had depicted the way she had perceived herself in the mirror during all those numerous attacks. The bright colours in the background, she explained to me, symbolised the element of chromatopsia that she had witnessed so often, with all the colours in her surroundings being extra-saturated and fluorescent. I did not dare to accept it as a gift; however, she insisted and said that she could hardly look at it

anyway because of all the bad memories it evoked. So I kept it in my consulting room for myself and others to see—and I proudly present it here (Fig. 3.5) as a token of Ms. Rembrandt’s artistic *and* diagnostic skills, as well as a reminder to myself, to go on listening to my patients, and work together with them in search of the correct diagnosis.⁵



Fig. 3.5 Self-portrait, acryl painting (2017) by my patient, Ms. Rembrandt; reproduced with kind permission. What Ms. Rembrandt expertly conveys here, is the way she felt (and saw herself in the mirror) during her ‘attacks’, which were probably of an epileptic nature

3.3.3 Visual Distortions , Part I: Macropsia and Micropsia

Back to Alice now, intimately connected with her sensation of feeling her body grow smaller and larger, is her perception of things appearing larger or smaller. This pair of symptoms is known as macropsia and micropsia. Just as Ms. Rembrandt sometimes saw the room becoming much larger than it was, in the story in a similar way, Alice sometimes sees the glass table as disproportionately large (when she herself is small), the door to the garden as disproportionately small (when she herself is large), the creatures in Wonderland as either small or large and so on. In the end, she grows so large that the creatures in the Courtroom, and indeed everything around her, appear to be very small (Fig. 3.6). It is a very common pair of symptoms that is mentioned by many of my patients, and even by colleagues and acquaintances who tell me about brief instances experienced during a state of fever (without necessarily having the accompanying sensation that their body has altered in size).



Fig. 3.6 Alice Upsetting the Juror Box, illustration by Sir John Tenniel (1890)

3.3.4 Hyperschematia and Hyposchematia

Another pair of symptoms intimately connected with the sensation of growing larger and smaller is the sensation of occupying more or less space; Todd referred to this pair of symptoms as hyperschematia and hyposchematia, respectively. In the *Alice* book, we find wonderful examples of both sensations, for instance, when Alice is fanning herself and then drops the fan hastily, ‘... just in time to save herself from shrinking away altogether’:

‘That was a narrow escape!’ said Alice, a good deal frightened at the sudden change, but very glad to find herself still in existence [9].

She here experiences *hyposchematia*, or the sensation of occupying very little space, in this case up to the point that she fears that she will shrink away altogether. An example of the opposite condition, *hyperschematia* (Fig. 3.7) can be found in the scene where Alice grows so large that she gets stuck in the White Rabbit’s house:



Fig. 3.7 Alice Stuck in the White Rabbit’s House, illustration by Sir John Tenniel (1865)

...before she had drunk half the bottle she found her head pressing against the ceiling, and had to stoop to save her neck from being broken... She went on growing, and growing, and very soon had to kneel down on the floor: in another minute there was not even room for this, and she tried the effect of lying down with one elbow against the door, and the other arm curled round her head. Still she went on growing, and, as a last resource, she put one arm out the

window, and one foot up the chimney, and said to herself, ‘Now I can do no more, whatever happens. What will become of me?’ Luckily for Alice the little magic bottle had now had its full effect, and she grew no larger: still it was very uncomfortable... [9]

3.3.5 Psychosomatic Duality

A fifth symptom described in the *Alice* book, one that we also encountered before, is psychosomatic duality. While Alice sits down reprimanding herself for crying—after having tired herself out with her fruitless attempts to climb up the legs of the glass table—Dodgson propels the story on as follows:

‘Come, there’s no use in crying like that!’ said Alice to herself, rather sharply. ‘I advise you to leave off this minute!’ She generally gave herself very good advice (though she very seldom followed it), and sometimes she scolded herself so severely as to bring tears into her eyes; and once she remembered trying to box her own ears for having cheated herself in a game of croquet she was playing against herself, for this curious child was very fond of pretending to be two people. ‘But it’s no use now,’ thought poor Alice, ‘to pretend to be two people! Why, there’s hardly enough of me to make one respectable person!’ [9]

As Dodgson tells us, pretending to be two people was one of Alice’s favourite pastimes. The *as if* character of the situation is important because psychosomatic duality, as we saw, is not about a ‘split’ personality or about multiple personalities taking turns at controlling the ego at different moments in time. Rather, it is about the transient sensation of being ‘split’, in the here and now, as exemplified by the patients described by Todd, who experienced having a second head or a shadowy ‘double’ that contained their mind. The exact way in which this psychosomatic duality can be experienced differs for different people, but the common theme is a fracturing of one’s sense of unity, and the subsequent confusion arising from having to deal with oneself from the vantage point of two locations in space.

Years ago, I treated a man in his 40s who told me beamingly how he would walk the streets at night, experiencing a double attached to one side

of his body who would mimic his every movement, and walk perfectly in step with him whenever he strolled, ran or even danced along. Meanwhile, he would be looking at the double's laughing face and exchange jokes that cracked them both up with laughter while they ran. Although he realised that all the jokes stemmed from his own mind, the sensation thus created was that his mind resided in two heads, and that these two heads worked in perfect harmony with each other to increase the fun. As we saw, in Alice's case, the locus of control remains in one place. In contrast to this patient, she does not experience herself as operating from two different locations simultaneously; however, what she goes through comes close to it, as she does communicate with herself in the here and now, *as if* she were two people.

3.3.6 Visual Distortions, Part II: Prosopometamorphopsia

A sixth symptom described by Dodgson is prosopometamorphopsia, the apparent change of faces into different ones, or, in a milder form, the apparent distortion of facial features. When Alice finds herself in the smoky, peppery atmosphere of the kitchen where the Duchess and her cook throw kitchen utensils at every other person in the room, she catches the howling baby flung to her and tries to hold it in her arms. What—until then—had seemed to be a rather ordinary baby, now suddenly strikes her as a queer-shaped little creature, 'just like a starfish'. And yet it is still a baby, even though it snorts like a steam engine in the harsh environment of the Duchess' kitchen, doubles itself up and straightens itself out in her arms, making it difficult for her to hold on to it. Once taken outside into the open air, the little thing starts to grunt. When Alice looks into its face to see what is wrong, she notices that the baby has a '... very turn-up nose, much more like a snout than a real nose'. What is more, its eyes become extremely small for a baby's and, altogether, Alice does not like its look at all. Telling herself that the baby may perhaps have been sobbing, she looks again. Seeing no tears, she then warns it that it must not turn into a pig or she will have nothing more to do with it. The way things go in Wonderland, the baby does of course turn into a pig (Fig. 3.8), upon which Alice sets it down and feels quite relieved to see it off, trotting away into the wood.



Fig. 3.8 Alice Carrying a Pig, illustration by Sir John Tenniel (1890)

What is interesting about this scene, is that there is not only a transformation from a human face to a pig's face but that, initially, the human face remains what it is for a short period of time. We already met Mrs. van Nuys, who saw human faces change gradually into dragon faces and, she too, reported that for the first few minutes she would have a stable

image of the person's actual face. Such a latency period preceding the moment of visual distortion is historically considered a sign of *cerebral asthenopia*, which is a fancy term for saying that there is an unusual fatigability of the perceptual system [10, 11]. The concept is based on the observation that, in such cases, the visual system is apparently capable of generating a faithful representation (of a face, in these examples), but after a while loses that ability and, instead, settles for a distorted image, which is then upheld for the remainder of the time. Although the exact mechanism causing this delay remains to be elucidated, the concept of cerebral asthenopia suggests that it must be some kind of exhaustion at the level of neuron populations in the visual system.

Another patient of mine, Willem, a gifted artist in his early 20s, told me that he often sees his own face in the mirror as if it were indented. This peculiar deformation always affects the left side of his face, just below the eye. The part of the cheek that he thus sees missing has a rectangular shape the size of a brick, as if he were a cartoon character that had just been hit hard in the face and is therefore left with a brick-shaped indentation. Once he perceives himself this way in the mirror, he also often sees his left eye protruding, and when he keeps on looking, he may see it gently popping out of its socket, descending slowly along the part of the cheek that is still there. Obviously, Willem has never been happy to see his face morph like this, even though he only sees it happening in the mirror—and touching his face teaches him that it is really intact. Before he came to see me at the outpatient clinic, he had had no idea why this occurred to him, wondering for many years whether he had gone crazy or something. Nevertheless, he had succeeded in making a virtue of necessity because the first time I saw him, he showed me a notepad, which he always carried with him for doodling and sketching; it contained several of his comics, with a main character whose head was indented exactly the way Willem saw his own during his attacks.

A difference between this type of prosopometamorphopsia and the kinds experienced by Mrs. van Nuys and Alice (in the story), is that Willem (like Ms. Rembrandt) sees his own face change, rather than other people's faces. Another difference is that his face remains recognisably his own. Even though it takes on grotesque proportions, with the brick-shaped dent in the cheek and the left eye slithering over the cheekbone, it is still *his* face, whereas in the first two cases, the faces perceived are gradually

robbed of their human features and are replaced by animal faces. Despite these phenomenological differences, both types of visual distortion fall in the category of prosopometamorphopsia.

3.3.7 Visual Distortions, Part III: Loss of Stereoscopic Vision

In the *Alice* book, a seventh symptom can be discerned in the scenes with the pack of cards, towards the end of the story. When Alice finally succeeds in entering the beautiful garden and finds herself among the bright flower beds and the cool fountains for which she had so long craved, she soon meets three gardeners busily painting the white roses red (Fig. 3.9). After she has made their acquaintance and asked them why they are painting them (which, you may remember, is to cover up the mistake they had made by planting a white rose tree instead of a red one), there is a sound of many footsteps. Looking around, Alice then spots ten soldiers carrying clubs, ‘all shaped like the three gardeners, oblong and flat, with their hands and feet at the corners’. As we soon learn, they are playing cards, which land flat on the ground when they bow for the King and Queen of Hearts . Seeing persons such as these gardeners, or any other three-dimensional objects, as ‘oblong and flat’ is what we call *loss of stereoscopic vision*.

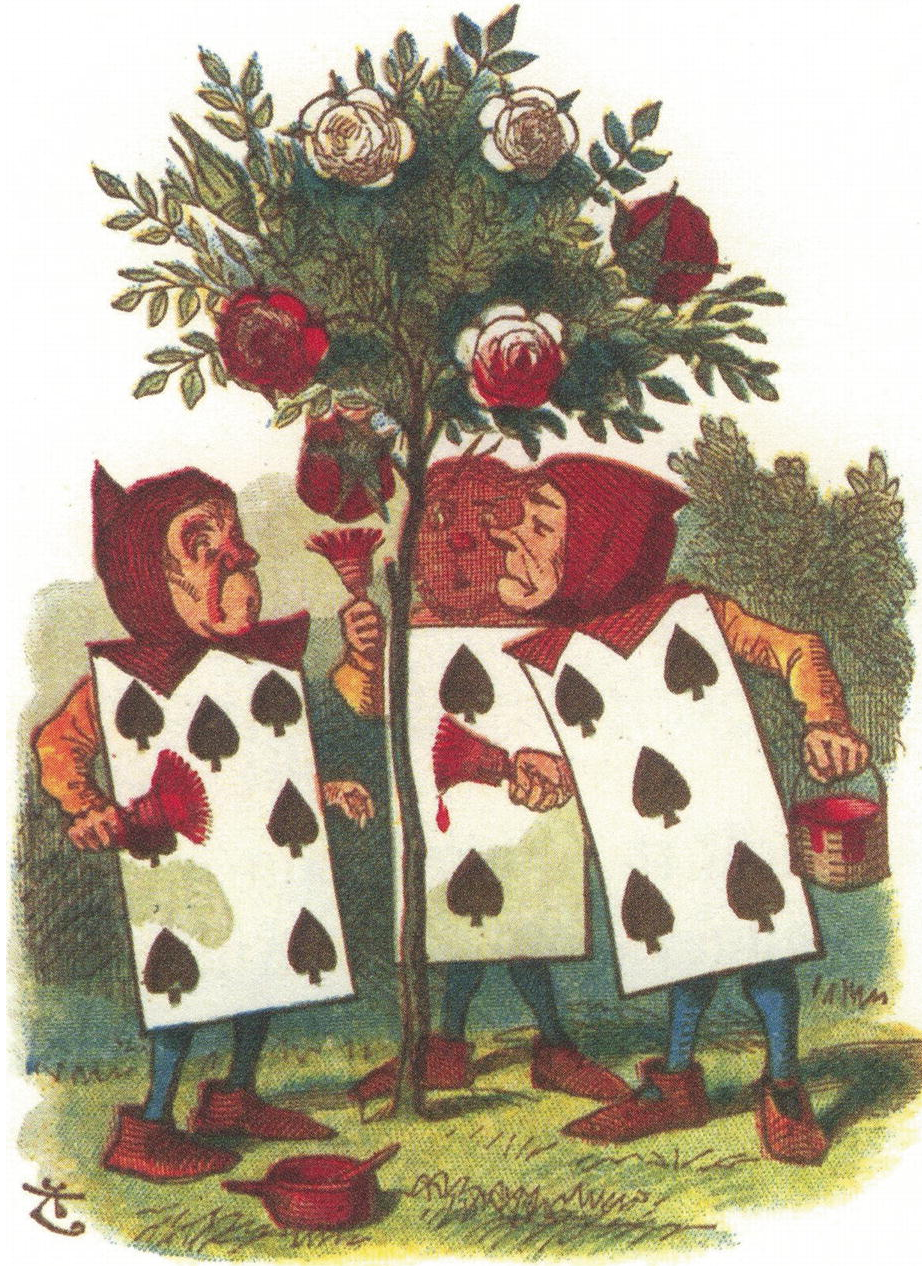


Fig. 3.9 Cards Painting the Roses Red, illustration by Sir John Tenniel (1890)

For stereoscopic vision to occur in our everyday, waking lives, two sources of visual input are needed, each providing a slightly different angle on the source material. The brain then uses the subtle differences between the two input pictures to create the illusion of a third dimension, which we call depth. With only one eye, no stereoscopic vision can be attained. On the other hand, in dreams, where there is no actual input from two eyes (or from the eyes whatsoever), we may also perceive depth. This proves that

stereoscopic vision is indeed a trick of the brain. Yet seeing with two eyes while we are awake does not necessarily guarantee that the brain will always perform this trick. It may just shut down its 3D function, which is intimately connected with a group of neurons known as the *binocular depth cells*. This, similarly, leads to a loss of stereoscopic vision, of which the gardeners and soldiers, and the King and Queen of Hearts in the *Alice* story, are perfect examples. In real life, a person experiencing loss of stereoscopic vision will obviously perceive *everything* as oblong and flat, rather than individual objects; however, that does not discount the beauty of the description provided by Dodgson in the book.

3.3.8 Time Distortions

An eighth symptom described in the *Alice* book is time distortion. Time is a recurring theme throughout anyway, starting with the White Rabbit on page one, which says to itself, ‘Oh dear! Oh dear! I shall be too late!’ and goes on to take a watch out of its waistcoat pocket and look at it before it hurries on. When Alice later finds herself trapped in the White Rabbit’s house, having grown so large that she is stuck in one of the rooms, she ponders on how it might be to never get any older than she is. On the one hand, she would find it a comfort, as she would then never have to be an old woman, although, on the other, she would hate to stay the age at which she would always have to learn lessons. After she has freed herself and has done some walking about, she finds herself in the company of the Duchess and gets all philosophical with her about time:

‘If everybody minded their own business,’ the Duchess said in a hoarse growl, ‘the world would go round a deal faster than it does.’

‘Which would not be an advantage,’ said Alice, who felt very glad to get an opportunity of showing off a little of her knowledge. ‘Just think what work it would make with the day and night! You see, the earth takes twenty-four hours to turn round on its axis-----’

‘Talking of axes,’ said the Duchess, ‘chop off her head!’

Alice glanced rather anxiously at the cook, to see if she meant to take the hint; but the cook was busily stirring the soup, and seemed

not to be listening, so she went on again: ‘Twenty-four hours, I think; or is it twelve? I----’ [9]

Dodgson himself was greatly intrigued with time and throughout his life sought to solve the riddle of what would happen if one would travel at the very speed at which the world turns on its axis, keeping pace with the rotation of the earth, and thus arriving at the same time in subsequent places, until one would be back where one began. He had already asked himself this question as a child and, apparently, it fascinated him so much that as an adult he kept repeating the question, in his diary, in pamphlets he published, and in front of audiences while lecturing about the subject, asking himself and his audience whether one would then still find oneself at the same time of the same day, or whether somewhere a day would have been lost. This problem was eventually solved in 1884 with the establishment of the International Date Line, an imaginary line of navigation that runs around the anti-meridian at 180° line of longitude, from the North Pole to the South Pole, to provide an artificial demarcation for the change of one day into the next one. This example from real life illustrates that Dodgson did not take the subject of time for granted. Perhaps because of that, he referred to it so often in the *Alice* book. Or could it have been because he had experienced time distortions himself? At any rate, like his creator, the Mad Hatter shows himself very much intrigued with time:

Alice sighed wearily. ‘I think you might do something better with the time,’ she said, ‘than wasting it in asking riddles that have no answers.’

‘If you knew Time as well as I do,’ said the Hatter, ‘you wouldn’t talk about wasting it. It’s him.’

‘I don’t know what you mean,’ said Alice.

‘Of course you don’t,’ the Hatter said, tossing his head contemptuously. ‘I dare say you never even spoke to Time!’

‘Perhaps not,’ Alice cautiously replied; ‘but I know I have to beat time when I learn music.’

‘Ah! That accounts for it,’ said the Hatter. ‘He wo’n’t stand beating. Now, if you only kept on good terms with him, he’d do almost anything you liked with the clock. For instance, suppose it were nine o’clock in the morning, just time to begin lessons: you’d only have to whisper a hint to Time, and round goes the clock in a twinkling! Half-past one, time for dinner!’ [9]

What the Hatter refers to in this passage is the speeding up of actual, chronological time . When people have the experience of that happening, it is called a quick-motion phenomenon , a term loosely translated from the German *Zeitrafferphänomen* , or ‘time-quickenening phenomenon’, as it translates literally. It refers to a type of time distortion in which psychological time (rather than actual, chronological time) is speeding up. In a previous section, I described the young man who had experienced the quick-motion phenomenon while walking from our hospital building to the bus stop, alternated by the opposite phenomenon of *protracted duration* , or a slowing-down of psychological time. We also met the patient who had been admitted to our hospital and experienced a total absence of the feeling of time. Luckily for the latter patient, after a few days, his sense of the passage of time returned. Since he had been admitted with a drug-induced psychosis and, by then, the effects of the cannabis had worn off, we attributed this temporary aberration to his substance use. The Mad Hatter refers to a similar phenomenon when he subsequently tells Alice that Time could be persuaded to ‘keep it to half-past one as long as you liked’, and goes on to say that, for him, ‘It’s always six o’clock now.’

Near the end of the book, Dodgson presents us with yet another time distortion . He does so quite implicitly, but when Alice wakes up with her head in her sister’s lap and tells her about her dream , we, the readers, know that she must feel as though she had been down in Wonderland for as long as a day or something, considering all the adventures she has had, and the time we ourselves have spent reading, whereas her sister cannot be expected to have been so patient to wait for her that long. If sisters manage to tolerate each other for an hour or so in each other’s lap, they are admirably patient sisters. Dodgson knew that, of course, explaining in a letter to a friend of his that indeed, ‘The heroine spends an hour underground, and meets various birds, beasts, etc.’ [12]. In other words, the

whole Wonderland story boils down to Alice experiencing a day's worth of adventures within the time frame of just 1 hour.

3.3.9 Derealisation and Depersonalisation

The ninth and final symptom—or, rather, pair of symptoms—is derealisation and depersonalisation. Throughout the story, Alice is almost constantly amazed at what is happening, and feels estranged by all that is going on. As we saw, that sense of estrangement, as if things were not quite real (which in Alice's case is exactly the point) is what we call derealisation. Depersonalisation, as we saw, is the sensation of not owning up to one's personal identity. That is what Alice experiences too, first and foremost in conjunction with the alterations of the size of her body and also when she reflects on those alterations and asks herself whether she is still herself, or whether she might perhaps have been changed into Ada or Mabel, two classmates of hers. While dealing with that sense of fundamental self-doubt, the Caterpillar does not exactly help her to find a firm sense of personal identity. Dodgson makes her share that feeling with us by confronting her with the Caterpillar's piercing questions about identity:

‘Who are you?’ said the Caterpillar.

This was not an encouraging opening for a conversation. Alice replied, rather shyly, ‘I - I hardly know, Sir, just at present - at least I know who I was when I got up this morning, but I think I must have changed several times since then.’

‘What do you mean by that?’ said the Caterpillar sternly. ‘Explain yourself!’

‘I ca’n’t explain myself, I’m afraid, Sir,’ said Alice, ‘because I’m not myself, you see.’

‘I don’t see,’ said the Caterpillar.

‘I’m afraid I ca’n’t put it more clearly,’ Alice replied very politely, ‘for I ca’n’t understand it myself to begin with; and being so many different sizes in a day is very confusing.’

‘It isn’t,’ said the Caterpillar.

‘Well, perhaps you haven’t found it so yet,’ said Alice; ‘but when you have to turn into a chrysalis - you will some day, you know - and then after that into a butterfly, I should think you’ll feel it a little queer, wo’n’t you?’

‘Not a bit,’ said the Caterpillar.

‘Well, perhaps your feelings may be different,’ said Alice; ‘all I know is, it would feel very queer to me.’

‘You!’ said the Caterpillar contemptuously. ‘Who are you?’

Which brought them back again to the beginning of the conversation. [9]

Dodgson here indicates, in this excellent depiction of depersonalisation , that Alice has undergone so many changes during the day that she does not know who she is anymore. It is important to note that this has nothing to do with her memory or cognition, because throughout the story she demonstrates convincingly enough that she remembers her name, that she knows where she lives, that she knows who her family members are, that she knows about Dinah, her cat, that she knows about school and that she is also totally capable of reflecting on all that. Moreover, she knows perfectly well what size and proportions she ought to have, and even remembers how she ended up in this awkward situation with the anal-sadistic Caterpillar . This is a wonderful example of depersonalisation . Yet the most evocative example in the *Alice* book is that of the Cheshire Cat , who—like Mr. Salvatore when he rides his bike—is the epitome of a disembodied self, disappearing from back to front until only his grin remains visible. ‘Well!’ is what Alice aptly remarks at the sight of it, ‘I’ve often seen a cat without a grin, but a grin without a cat! It’s the most curious thing I ever saw in all my life!’

3.3.10 Was It All a Dream?

As all these passages indicate, the *Alice* story contains quite a few references to perceptual distortions . Which brings us back to the question

of how they got there in the first place. Did Dodgson just make them up, the way he made up so many things? Or is there something more to them? Could he have dreamed them, for instance? Or could he have experienced them in the border area between waking and sleeping? *Alice's Adventures in Wonderland* is such a convoluted blast, devoid of any conventional plotting, that the idea of a dream sequence is not entirely out of place. However, judging by what Dodgson wrote about the story's genesis, it did not seem to stem from an actual dream, even though many elements do appear to have come from a place that only later was designated by Sigmund Freud (1856–1939) as 'the subconscious'. Dodgson indeed indicated that the subconscious might have had something to do with it, by saying that many of the ideas for his book had come 'of themselves', musing that he had had

... many fresh ideas, which seemed to grow of themselves upon the original stock; and many more added themselves when, years afterwards, I wrote it all out for publication; but every word of the dialogue came of itself. Sometimes an idea comes at night, when I have to get up and write it down - sometimes when out on a lonely winter walk, when I have had to stop, and with half frozen fingers, jot down a few words which would keep the new-born idea from perishing - but whenever and however it comes, it comes of itself.
[13]

This suggests that Dodgson might well have found himself a way to access the 'primary process,' as psychoanalysts call this mode of thinking. In an evolutionary and developmental sense, the primary process is the type of mental process that predates linguistic reasoning. It is considered more primitive in nature, in the sense that it is immediate, illogical and raw, and that it makes use of images, emotions, and loose associations rather than the carefully built concepts that we use when we express ourselves in language. In comparison with what Friedrich Nietzsche (1844–1900) called our Apollonian mode of thinking, which makes use of the soberness and precision of logic and linguistic expression, it stands for our creative, whimsical, excessive and sometimes even violent side: our Dionysian side, in short, to stick to Nietzsche's idiom. It would seem that Dodgson was indeed capable of accessing that layer of his consciousness, comparable to the way artists such as Salvador Dalí (1904–1989) would later tap into the

deeper layers of their psyche to retrieve images or ideas that would otherwise have remained beyond their reach [14]. This may sound a bit woolly, but Dalí went about this business with the primary process quite methodically, applying Freud's principles—of which he was an avid fan—by taking a nap while sitting in a chair, with one arm dangling down, and a spoon held loosely between his fingers. Each time that he would nod off in that position, the spoon would come rattling down on a tin plate that he had placed underneath for that specific purpose, waking him up instantly, and allowing him to catch a glimpse of what he had just begun to dream . Using the imagery thus brought into his consciousness is what lent many of his paintings their well-known surreal quality. Other artists have used hypnosis or self-hypnosis to achieve a similar effect, and still others used illicit substances . Although Dodgson does not seem to have belonged to the latter group, the images he evokes throughout the *Alice* story are nonetheless reminiscent of the LSD experience so, in that sense, it is not too far-fetched that some authors believe that he had actually been on psychotropic substances. This is partly due to the disjointed storyline and partly to the recurring theme of distorted perceptions.

Perhaps such elements just presented themselves to him without much conscious effort. As he had a habit of jotting down brain waves in the dark, we may assume that at least part of his inspiration stemmed from a similar source as Dalí's , even though the two of them used different methods to access it. For someone so adamant about generating new ideas, it should not surprise us if Dodgson also recorded hypnagogic or hypnopompic phenomena , meaning that he may have used imagery that presents itself during the phases of falling asleep and waking up. However, before we start speculating about all that, perhaps we should take a closer look at the man himself, at his medical history, and at the way the *Alice* book was written. With that, the narrative will take a completely different turn. But don't worry, after this small detour, we will soon be back in business about Alice in Wonderland syndrome itself.

References

1. Davis M (2013) West Riding Pauper Lunatic Asylum through time. Amberley Publishing, Gloucestershire
2. Davis M (2013) Voices from the asylum. West Riding Pauper Lunatic Asylum. Amberley

Publishing, Gloucestershire

3. Todd J (1955) The syndrome of Alice in Wonderland. *Can Med Assoc J* 73:701–704
[PubMed][PubMedCentral]
4. Coleman SM (1933) Misidentification and non-recognition. *J Ment Sci* 79:42–51
[Crossref]
5. Lippman CW (1952) Certain hallucinations peculiar to migraine. *J Nerv Ment Dis* 116:346–351
[Crossref]
6. Todd J (1954) Trichlorethylene poisoning with paranoid psychosis and Lilliputian hallucination. *Br Med J* 1:439–440
[Crossref]
7. Lejeune J, Turpin R, Gautier M (1959) Le mongolisme, premier exemple d’aberration autosomique humaine. *Ann Genet* 2:41–49
8. Frederiks JAM (1963) Macrosomatognosia and microsomatognosia. *Psychiatry Neurol Neurochir* 66:531–536
9. Carroll L (1866) *Alice’s adventures in Wonderland*. Macmillan & Co., London
10. Pötl O (1928) *Die optisch-agnostische Störungen*. F. Deuticke, Leipzig
11. Willanger R, Klee A (1966) Metamorphopsia and other visual disturbances with latency occurring in patients with diffuse cerebral lesions. *Acta Neurol Scand* 42:1–18
[Crossref]
12. Dodgson CL (1979) Letter to Tom Taylor, June 10, 1864. In: Cohen MN (ed) *The letters of Lewis Carroll*. Edited by Morton N. Cohen with the Assistance of Roger Lancelyn Green, vol 1, ca. 1837–1885. Oxford University Press, New York
13. Reed L (1932) *The life of Lewis Carroll*. W. & G. Foyle, London
14. Blom JD (2018) Hallucinaties en kunst. *Tijdschr Psychiatr* 60:37–45
[PubMed]

Footnotes

¹ The term nosologist comes from the Greek words *nosos* (disease) and *-logia* (study of). It refers to a person involved with the description and classification of diseases.

² Among the themes Todd addressed in his writings were autoscopy (meeting a hallucinated doppelgänger), Othello syndrome (a name newly coined by him to designate pathological sexual jealousy), folie à deux (shared delusional disorder) and Capgras’ syndrome (the inability to identify a known person, believing he or she has been replaced by a double).

3 While at Littlemore, Todd himself had described a patient who had experienced micropsia , that is, seeing things smaller than they are [6]. Interestingly, in that paper, he had asked himself whether Jonathan Swift (1667–1745), the author of *Gulliver's Travels* , might have suffered from micropsia or 'lilliputian hallucinations ' himself, the way he would later ask himself about Charles Dodgson and the symptoms considered characteristic of Alice in Wonderland syndrome.

4 After that conceptual breakthrough, Down's syndrome should have been renamed Down's disease , but apparently, the name had become so familiar that no one bothered to change it. However, strictly speaking, the term 'syndrome' is still reserved for more or less fixed configurations of signs and symptoms, the connecting element of which eludes us.

5 Obviously, the story of Ms. Rembrandt did not end there. After having enjoyed the first few weeks free of any attacks, life itself came back to her with a blow. Like the rest of us, she found herself confronted with the daily hassles of ordinary life, but, in her case, they were aggravated by the legacy of 40 years of misunderstandings, forced decisions, faltering relationships and missed opportunities. It was only at this point that Ms. Rembrandt could properly start working towards her recovery, and she had a long, long way ahead of her, even though she no longer had to deal with the recurring symptoms of Alice in Wonderland syndrome.

4. Charles Dodgson

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

One golden afternoon in 1862, two Oxford dons, clad in white boating suits with straw hats, were rowing up the River Thames, locally known as the Isis. With them, they had an unusual party of three little girls who were having a great time, taking turns at the oars, playing word games, exchanging jokes, and singing along with the dons, one of whom had a particularly good voice. Throughout their playful and pleasant journey on that blazing summer day, they kept begging for a story to be told. After a while, the other don gave in and started telling a curious tale, making it up as they went along. Although he usually had a slight stammer, in the company of the three little girls he spoke without a single hesitation. Entranced, the girls kept begging for more and each time he said, ‘*The rest next time*’, they cried out happily ‘*It is next time!*’, and then the don would add another curious episode; this continued all the way to Godstow and, after a leisurely tea break on the river bank, all the way back to Oxford, until finally the sun went down and the day was over, and with it the curious tale. Told on that single afternoon of July 4, 1862, and since immortalised in written form, this is the myth of how the story of *Alice’s Adventures in Wonderland* came to life.

The two Oxford dons were the Reverend Charles Lutwidge Dodgson (1832–1898), aka Lewis Carroll (Fig. 4.1), and his confrere Robinson Duckworth (1834–1911). The storyteller, of course, was Dodgson, the singer Duckworth. The three little girls were Lorina, Edith, and Alice

Liddell (Fig. 4.2), daughters of Dean Henry George Liddell (1855–1891; Fig. 4.3), the highest-ranking authority of Christ Church and, hence, the academic superior of the two dons; and of Lorina Hannah Liddell *née* Reeve (1826–1910; Fig. 4.4), Oxford’s most influential social celebrity. At the time, Dodgson was 30 years of age. Legend has it that he cooked up the *Alice* story within those few hours on the water; however, it is not even certain whether this particular ‘*golden afternoon*’ ever existed. Weather reports of July 4, 1862, indicate that there was too much cloud over Oxford that day, and with a mean temperature of 68 °F (i.e. 20 °C), there could hardly have been a heat haze of the magnitude suggested by Dodgson. In addition, the author’s diaries indicate that the story had been told more like a serial—broken off and picked up time and again on separate occasions—while correspondence of a later date between Alice Liddell and her sister Lorina raises doubt as to whether Duckworth had been present at all.¹



Fig. 4.1 Charles Dodgson around the time he met the Liddell family (c. 1856-1860), by unknown photographer



Fig. 4.2 Sisters Alice, Lorina, and Edith Liddell, with their brother Henry (aka Harry), photographed by Charles Dodgson (1860)

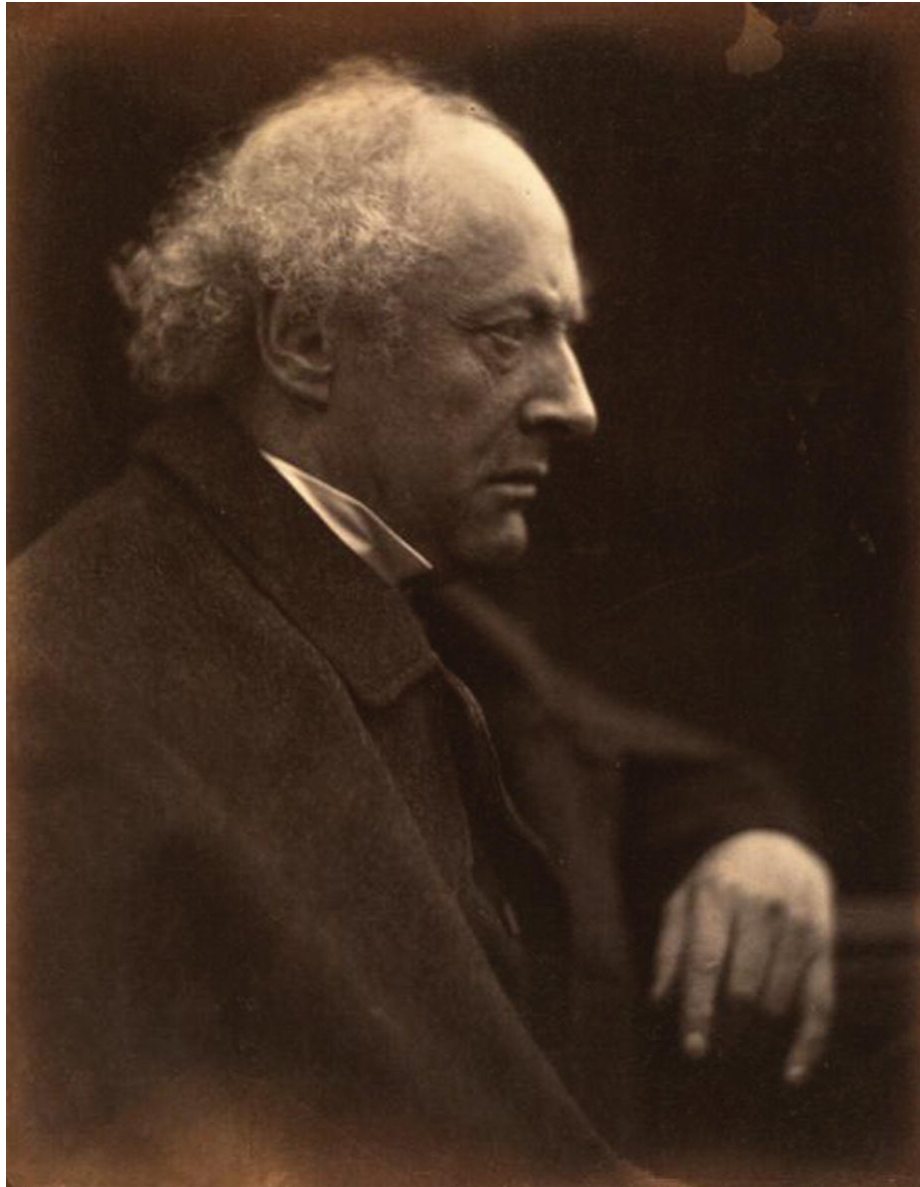


Fig. 4.3 Henry George Liddell, photographed by Julia Margaret Cameron (c. 1870)



Fig. 4.4 Lorina Hannah Liddell, née Reeve, by unknown photographer (c. 1860-1870)

What is certain, is that the story incorporated elements that had been brewing in Dodgson's mind for many years. As indicated by Thomas Fowler (1832–1904), he had already told stories about his beloved Alice to a circle of boys and girls at Whitby Beach as early as 1854, 8 years before the boating trip on the *Isis*, and more than a year before he had met the real Alice. Although all sources indicate that Alice Liddell had indeed requested to have the story written down on that memorable fourth of July of 1862, and Dodgson had set out to do so afterwards (either at night or during the following day, scribbling away on a train), this first heroic attempt only

allowed him to scratch the surface of the story as we know it today (Fig. 4.5). To write it down, and see it published in its final form, took him three long years, during which time he strayed from his path on numerous occasions. It is no wonder, therefore, that when the book came out in 1865, the story had undergone various transformations.

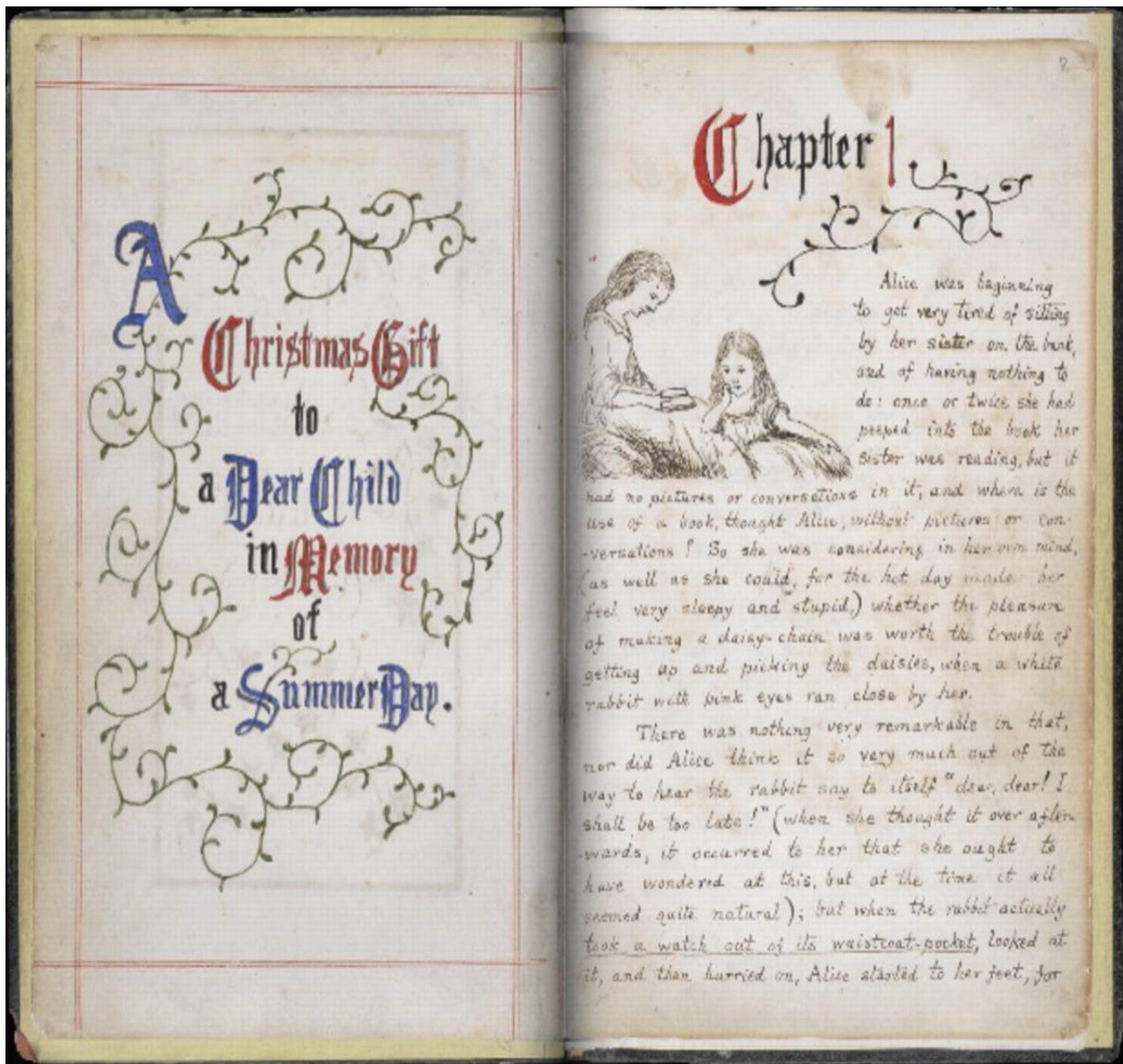


Fig. 4.5 Opening pages of *Alice's Adventures under Ground*, by Lewis Carroll (1862)

Its initial title, *Alice's Adventures under Ground*, had been dropped. The text had grown twice as long and new dialogues, verses, and plot twists had been added, whereas several others had been omitted. Owing to Dodgson's eye for detail, not to say his tendency to obsess over minutiae,

hammering out the final version had been a painstakingly slow process, drawn out even longer because the author was not easily pleased with the work of his illustrator, Sir John Tenniel (1820–1914; Fig. 4.6).² Even though, at that time, Dodgson himself was still unknown as a writer, Tenniel was a celebrity, an artist in great demand who catered to discerning audiences and held a position as leading cartoonist for *Punch*. Being accustomed to the whims of his commissioners, he was initially unruffled by Dodgson's relentless stream of highly detailed instructions about what he did and did not want to be included in the drawings. However, the way that Tenniel was put on the grill was more than even this seasoned illustrator could handle. At some point, he threatened to abandon the project altogether, so it is surprising that Dodgson did eventually get what he wanted—without losing Tenniel .³

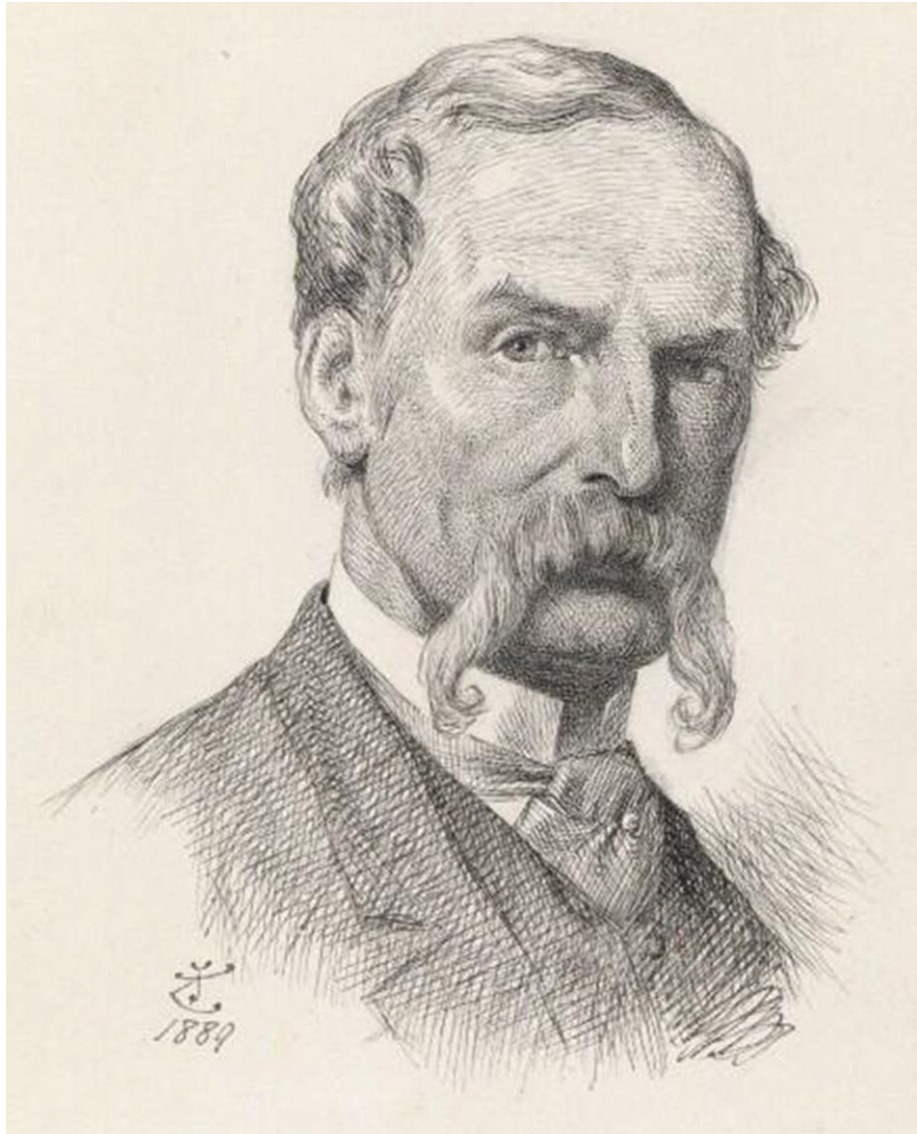


Fig. 4.6 Self-portrait of Sir John Tenniel, pen and ink on cream wove paper (1889)

In May, 1865, the book, now complete with illustrations and all, finally rolled off the presses at Oxford's Clarendon Press. Dodgson was so happy with the result that he immediately sent out presentation copies. However, the festive mood was soon nipped in the bud. In retribution, perhaps, for the way he had been treated, Tenniel showed himself displeased with the way the printing of the pictures had turned out. Dodgson, never one to countenance a glitch, even if it were merely in the eye of the beholder, briskly decided that no British child should be exposed to such an abomination. He called back all presentation copies and shipped off what

was left of the first batch of 2000 to the USA, minus 34 copies, which he sent to infirmaries and children's hospitals in the UK.

To his relief, the second run, printed at Clay's in the autumn of 1865,⁴ was entirely to Tenniel's—and his own—satisfaction. In his diary, he accordingly declared that this new impression was '*very far superior to the old, and in fact a perfect piece of artistic printing*' [3]. Thus satisfied, three and a half years after the moment that Alice Liddell had persuaded him to write the story down, he was able to offer her a first copy of the authorised version, exclusively bound for her in white vellum and adorned with a personal dedication.⁵

The rest, as they say, is history. With the exception of a few sour grapes, *Alice's Adventures in Wonderland* met with raving reviews in the press, and soon nestled itself in the top tiers of lists of popular children's books. Having done a bit of market research among acquaintances before deciding on a public release, Dodgson already had considerable confidence that his book would go down well with children—and yet neither he, nor anyone else, could have foreseen its dazzling success. His diaries indicate how he tracked sales figures and newspaper notices, and how he reacted to them with amazement and scarcely veiled pride—veiled, that is, because his character prohibited him from indulging in something as loathsome as pride. But pride it was, and for entirely justifiable reasons.

The source of that unexpected success has been the subject of ongoing scrutiny and speculation. Obviously, Dodgson had a way with words, and even though his exacting nature may have postponed the finalisation of his project, it certainly did not harm the story itself. A comparison with *Alice's Adventures under Ground*, which he had presented to Alice Liddell on Christmas Day, 1862—handwritten, with preliminary illustrations of his own—shows that he had skilfully ironed out all minor flaws, leaving out tiny bits that rang untrue to the story's overall spirit of nonsensical joy, and making all allusions to aggression (a nasty beast to tackle when you have a Queen of Hearts going, '*Off with their heads!*' at every occasion) palatable to the sensitive souls of children and the even more sensitive souls of their parents. On top of that, Dodgson was a master at parodying popular verses and poems—and including some of those parodies in his book was an act of sheer brilliance. Tenniel's illustrations, meanwhile, were indeed superior to his own attempts at drawing, lending the book a timelessly attractive visual style.

Nevertheless, that does not fully explain how *Alice's Adventures in Wonderland* could have become such a gem. Therefore, many believe that his relationship with the Liddell sisters had something to do with it. After all, they were the ones to whom the story had been told before he had committed it to paper, and they had also been the ones who had been allowed to play parts in it, albeit in a bit of a disguise. Dodgson never concealed the fact that he had been very fond of them and, perhaps, most of all of Alice, whom he afterwards lovingly considered the first in a long line of child friends, who meant more to him than any adult friend ever could. As he wrote later, on the occasion of the release of the facsimile edition of the book,

I distinctly remember, now as I write, how, in a desperate attempt to strike out some new line of fairy-lore, I had sent my heroine straight down a rabbit-hole, to begin with, without the least idea what was to happen afterwards. And so, to please a child I loved (I don't remember any other motive), I printed in manuscript, and illustrated with my own crude designs - designs that rebelled against every law of Anatomy or Art (for I had never had a lesson in drawing) - the book which I have just had published in facsimile [2].

Apparently, his little muse—or all three sisters together—had inspired him to channel an energy so creative and pure, so boundless and yet constrained, that the *Alice* story flowed from his pen, carrying with it dialogues and puns and images that washed up on the shores of his consciousness as if originating from a different place. Even though that did not happen during a single outburst of creativity, or an epiphany of sorts, Dodgson was able to revisit that well of inspiration time and again and, with his unerring sense for originality and wit, kept doing so until every piece had fallen into its proper place.

At what point Dodgson had become so impressed with Alice Liddell is unsure. It is not even sure when and how they met. Alice, who had been only 3 years old when she came to Oxford, was later unable to recall the moment [4]. In his diary of 1855, we see Dodgson himself circling the Liddell family as if he were already dimly aware of the prize lying in wait for him. The first Liddell child he mentions is a relative of Alice's, called Frederica (1848–1891), described by him in no uncertain terms:

Each time I see [Frederica Liddell] confirms me in the impression that she is one of the most lovely children I ever saw, gentle and innocent looking, not an inanimate doll-beauty [5].

That Dodgson considered Frederica '*not an inanimate doll-beauty*' was especially telling, since he had a refined sense for authenticity, and detested mere superficial looks and manners. On September 4, he added that she was '*one of the nicest children I have ever seen, as well as the prettiest*', before exclaiming, '*Dear, sweet, pretty little Frederica!*' [6].

Next up was Frederica's younger sister, Gertrude , whom he considered '*even prettier than my favourite Freddie*'. After meeting Gertrude, it would take six more months before the Dean's own children came into view, but then, one day at the Torpids, he met Alice's brother, Henry (aka Harry), and Lorina , their sister. He soon '*made friends*' with them and called Harry '*certainly the handsomest boy I ever saw*' [7, 8]. It is not inconceivable, however, that he had already spotted the kids some time before, as the previous year he had obtained the post of sub-librarian of Christ Church , and in that capacity had gained access to a room looking down on the Deanery garden, where he must have been able to see them play, together perhaps, with the Dean and his wife, the notoriously beautiful Lorina Liddell , whose good looks had apparently passed on to her children. Nonetheless, Alice Liddell only appears in the diaries over a year later, on April 25, 1856. In that entry, Dodgson describes how he had gone over to the Deanery to take a photograph of the Cathedral and had attempted to make the children pose in the foreground. Even though he later complained that they were '*not patient sitters*', a photograph does survive (Fig. 4.7), and he himself marked that day '*with a white stone*', a phrase borrowed from the Roman poet Gaius Valerius Catullus (ca. 84–54 BC) to mark the days on which he had felt particularly happy or content.



Fig. 4.7 *Lorina, Alice, and Edith Liddell in the Deanery garden, photographed by Charles Dodgson (1856)*

As unusual then as it is now, Dodgson quickly succeeded in building a friendship with Alice Liddell and her siblings, and started visiting the Deanery on a regular basis. He even obtained permission to take the children with him, reading stories to them in his rooms, playing backgammon with them, letting them solve puzzles and riddles, guiding them through logical and mathematical problems, taking them to the boat races and visits of members of the Royal Family, taking them out rowing, playing *croquet* with them, photographing them, and allowing them inside his darkroom, that magical realm of the industrial age where the children could see their own faces gradually take shape in mysterious acid baths, rocked gently by their illustrious friend. In his diary, Dodgson characteristically marked such days with a white stone. Sometimes the children were escorted by their governess, Miss Mary Prickett (1832–1920), but at other times, he had them all to himself.⁶ Before long, ties with the family had become so close that when the Dean spent the winter months of 1856–1857 away in Madeira for the sake of his health, taking with him only his wife, the two of them left Alice and her siblings behind in Oxford, with Miss Prickett to watch over them, and Dodgson as an unofficial parent

figure in the background. During those months, Dodgson became an even more regular visitor to the Deanery, setting up his camera and dark room over there without bothering to take them home between sessions and photographing the children and numerous visitors, whom he invited over to the Deanery as often as he liked.

Although all of this happened under the watchful eye of Ms. Prickett, who in her position as governess kept the children's mother informed by letter, after a while rumours began to spread that Dodgson's intentions with the Liddell household might not be as pure as he cared to admit. After the Dean and his wife had returned, Dodgson wrote in his diary that his '*notice of [the children] is construed by some men into attentions to the governess*', although other insinuations may perhaps also have circulated. Upon hearing this, he resolved to refrain from taking any public notice of the children; however, that was easier said than done. The unusual friendship meant so much to him that within 10 days he was back in their company again and, from then on, continued to see them as regularly as before. As his diaries indicate, there were times that he saw them every day of the week. In fact, he became such an integral part of the family that he came along on visits to the grandparents and was trusted to take the children under his wing when the Dean and his wife were occupied by family circumstances. With the Dean himself probably being a rather absent and detached figure in the Liddell household, due to his governing function at Christ Church, the continuing work on his Greek lexicon, and all the other things that must have been on his mind, there had been a parental void to fill. Perhaps because of that, or perhaps because the Dean's wife, Lorina Liddell, had a busy social schedule of her own, or for reasons that we have no idea of, Dodgson was allowed to fill that vacant space and act as a quasi-parental figure for the children. He fulfilled that function for over a year, and possibly longer; unfortunately, because his diary volumes from April 1858 through May 1862 are missing, we cannot be sure how long the situation actually lasted. But even a year is long for a man to walk in and out of another man's house, to spend time with his wife and children on an almost daily basis, to be seen in public occupying his place, and to radiate an obvious sense of warmth and charm at the sheer excitement of being in such a privileged position. If that was not curious enough, the situation was all the more remarkable since, at work, Dodgson was engaged in a trench war with the Dean, debating his plans to modernise the university, criticising his

proposals and decisions in public meetings, and satirising him in pamphlets and open letters. No wonder that Oxford took notice, and no wonder that Oxford gossiped.

Due to a missing page in the diary covering the days of June 27–29, 1863, it is unclear what happened next; nevertheless, somehow, Dodgson's relation with the Liddells went sour. It is not unthinkable that Lorina Liddell had also caught wind of the rumours, or that she fostered suspicions of her own. Or perhaps the Dean had finally decided to bring a halt to this whole absurd situation. For whatever it is worth, Dodgson's niece, Violet Dodgson (1878–1966), later produced a succinct summary of what the missing page had allegedly contained, stating that,

L.C. learns from Mrs. Liddell that he is supposed to be using the children as a means of paying court to the governess - He is also supposed soon to be courting Ina [9].

In this passage, '*L.C.*' undoubtedly stands for 'Lewis Carroll', while '*Ina*' may refer to Alice's sister as well as her mother, whose names were both Lorina. However, the addition that '*Ina*' was supposed to be courted '*soon*', suggests that this may have been about the sister, who in 1863 had turned 14, the age at which a girl became eligible for marriage in Victorian England. Whether this brief statement truly sums up what had been written on the missing page is unknown, so perhaps the safest way to deal with it is to consider it as just another piece of gossip. Nevertheless, whatever had been at issue, from then onwards, the carefree rendezvous were over. Dodgson remained aloof for half a year and then resumed his visits to the Deanery, although much less frequently. A few months later, he was told by Mrs. Liddell in no uncertain terms that there would be no more boat trips with the children, now or ever [10]. Although he characteristically refrained from dwelling on this development in his diary, it must have been a grave disappointment to him to be deprived so suddenly of the cherished privilege of being so close to Alice and her siblings, and to be barred from the household that he had been visiting for so long, and under such uniquely happy circumstances.

As the years went by, and the children grew up, meetings with them grew sparser and sparser, and finally there came years without any sort of communication. Nonetheless, Dodgson attempted to stay in touch with the

family throughout his life, providing Lorina and her children with inscribed presentation copies of his latest works—which he kept doing until a few years before his death—and receiving Alice Hargreaves *née* Liddell as late as 1891 in his Oxford studio, when he was 59 years and she 39 [11]. Although this may sound as if they both still cherished the special bond they had once had, it is not entirely clear how the meeting evolved and how his continuing attempts at contact were appreciated by her. After all, it must have been an embarrassing and confusing situation, with Dodgson disputing the Dean so bitterly and for so long on one front, satirising him in pamphlets, newspapers, and other media outlets, and lampooning him *and* his family in biting works of fiction, while, on the other hand, aspiring to restore the intimacy of bygone times, dedicating new products of the pen to Alice Liddell, offering presentation copies, and sending out letters that feigned love untainted.

Although in the meantime Dodgson had gone on to make friends with literally hundreds of other girls, he appears to have remained fascinated with Alice Liddell, or at least with her name, and the fictional character built around her persona, since he always showed himself delighted when he made the acquaintance of pretty girls whose name also happened to be Alice. Winning little girls—and sometimes boys—over with his wire puzzle and other playthings, which he always seemed to carry with him for that particular purpose, became a trademark strategy of his that he would successfully deploy for the remainder of his life. However, revealing his identity as the author of *Alice's Adventures in Wonderland* was a trick that he liked to keep up his sleeve until the moment when the first meeting with a new child threatened to come to an end. That was the premeditated point at which he would present his credentials as an Oxford don to the child's parents, or write them a note to ask permission to get to know their child. Often succeeding in thus obtaining the parents' consent, as well as the unsuspecting child's address, he would then send a copy of his book through the mail. Sometimes these potential child friends would only guess his true identity after they had opened the package delivered to their doors, and if the ice had not been broken by then, the book itself was bound to do the rest. Because by that time it had become such a phenomenon, that the fictional Alice had taken on a life of her own, and her creator the proportions of a saint.

4.1 The *Alice* Book and Its Real-World Connections

At first glance, *Alice's Adventures in Wonderland* may strike one as a bizarre and utterly nonsensical work of fiction. That feeling is heightened by the tangled storyline even though, structurally, it is relatively straightforward. As a frame narrative, it begins with Alice sitting in the grass near the bank of a pool, being tired of having nothing to do. Even her sister's book fails to rouse her interest, as it has '*no pictures or conversations in it*'. Cue the White Rabbit, a tumble down the rabbit hole (which, interestingly, does have pictures on its walls), and Alice dozing off during the rather lengthy descent, dreaming that she walks hand-in-hand with her cat, Dinah, until she lands *thump! thump!* upon a heap of sticks and dry leaves. And on goes the story, with Alice following the White Rabbit and meeting all the creatures in the curious Wonderland that we know so well (meanwhile having numerous conversations with them, which she also missed so much in her sister's book). When, finally, she is threatened by the Red Queen, and a pack of cards come flying down upon her (Fig. 4.8), she gives a little scream and finds herself back on the river bank, lying there with her head in the lap of her sister, who gently brushes away some dead leaves from her face. '*Wake up Alice dear,*' she hears her say, '*Why, what a long sleep you've had!*'—to which Alice replies, '*Oh, I've had such a curious dream !*'



Fig. 4.8 *The Shower of Cards*, illustration by Sir John Tenniel (1890)

With these opening and closing remarks, the story is presented as a dream sequence, experienced by the fictitious 7-year-old Alice who, in the context of the story's 'reality', does nothing but lie in the grass, nod off, and wake up in her sister's lap. Thus, her body remains near the bank of the pool, while her spirit ventures forth and witnesses the marvels of Wonderland. With this set-up, Dodgson revisits the oneiric dimension of the classical narrative model that allows the protagonist to have a vision during sleep (*visio in somnio* [12]) and thus places the story in a literary tradition

exemplified by Dante's *Divine Comedy* [13] , which begins with Dante Alighieri (1265–1321) falling asleep at the beginning of his journey and ends with him waking up spontaneously afterwards.⁷ However, in Alice's case, the transition to sleep is expertly concealed by the suggestion that throughout the story her stream of consciousness is uninterrupted, except the brief lapse of time during the tumble down the rabbit hole, when '*she felt that she was dozing off, and had just begun to dream*'. That sequence ends with the touchdown on a heap of dry sticks and leaves, and in what follows, Alice jumps to her feet, and spots the White Rabbit hurrying down a long passage (Fig. 4.9), which suggests that not only is the fall over, but also the dream; however, we are not explicitly told that this is the case—and for good reasons, because concealing Alice's dream state was exactly what Dodgson was after when he wrote the story. '*The whole thing is a dream*', he wrote to Tom Taylor (1817–1880), '*but that I don't want revealed till the end*' [15].



Fig. 4.9 *Alice and the White Rabbit Running*, illustration by Sir John Tenniel (1890)

As the story was meant to be nonsensical in nature, contrary to the majority of children's books in Victorian England—which were supposed to be educational in nature—we might be forgiven for assuming that it should not really matter what Dodgson had written down. In nonsensical books—as in dreams—anything goes, after all. And even if dream experts tell us otherwise, pointing out that dreams have their own inherent structure and logic, for nonsensical stories it is certainly true that anything goes. So why bother with the contents of the *Alice* story? And why bother with the nature of the perceptual phenomena described in it, if they can all be dismissed as stemming from the author's fondness of literary nonsense?

To find a preliminary answer to those questions, we only need to realise that Dodgson had dotted the *Alice* story with references to real life, beginning with the central character, of course, whose name and persona he had borrowed from Alice Liddell , and on whose birthday, the fourth of May, the story was said to take place. Alice's two sisters were given smaller roles—cameos one would say today—in the short story told by the Dormouse , where they appear as Elsie (i.e. 'L.C.', for Lorina Charlotte) and Tillie (for Matilda), while the name of the third character, Lacie, was an anagram for 'Alice' [4]. The White Rabbit may well exemplify a story-telling device called a psychopomp , or a guide of souls, who in classical mythology had the task of guiding recently deceased souls—and sometimes dreamers—into the underworld [14]. Considering the White Rabbit's preoccupation with time, in this incarnation, he may well have stood for the girls' father, the Dean, of whom it was said that he was always running late, although another contender for the true identity of the White Rabbit is Dr. Henry Wentworth Acland (1815–1900), the Liddells's family physician, who allegedly had a habit of regularly looking at his pocket watch, and, due to his function in public sanitation, was required to descend from time to time into Oxford's underground sewage systems [14]. Dinah, the cat, was modelled after a real cat named Dinah that lived with the Liddell's and was allegedly chased out of the Christ Church Library on more than one occasion [4]. A second cat owned by the Liddells was Villikins, the two of them being named after a popular comic song called *Villikins and his Dinah* [14]. The Duck featuring in the Caucus-race is said to stand for Duckworth , who may or may not have been there in the boat when Dodgson first told the story, whereas the Dodo almost certainly represents Dodgson himself, with a self-conscious riff on the author's speech impediment (Fig. 4.10) [4].



Fig. 4.10 *Dodo Handing Alice a Thimble*, illustration by Sir John Tenniel (1890)

The origin of these characters is quite certain, whereas others cannot be so easily traced back to their real-life sources—although it has been speculated that Bill the Lizard, who gets kicked out of the chimney when Alice finds herself stuck in the house of the White Rabbit, may have been a reference to Benjamin Disraeli (1804–1881), the politician who would later become Britain’s Prime Minister. Similarly, it has been suggested that the Cheshire Cat stood for Edward Bouvery Pusey (1800–1882), Dodgson’s patron and mentor at Christ Church, the Gryphon for John Ruskin (1819–1900), and the Mock Turtle for Dodgson’s friend and travel companion to Russia, Henry Liddon (1829–1890) [14]. The character of the Mad Hatter may well have been inspired by a fellow student of Dodgson’s at Christ Church [16] or, according to others, a waiter at Christ Church, who may or may not have been the same as Mr. Theophilus Carter (1824–1904), a cabinetmaker who later ran a furniture and upholstery store in Oxford.

Because of the latter's idiosyncratic ideas, his remarkable facial features, and his inseparable top hat (allegedly worn at the back of the head), Carter was an easy target for mockery and was called '*the mad hatter*' by the local community [4, 17]. It has been suggested that at some point Dodgson invited Tenniel over to Oxford to see the man, hoping that the illustrator would agree to portray the fictional Mad Hatter in his likeness. Tenniel, opposed to using models for his drawings, may nevertheless have taken a long hard look, sighed, and drawn him from memory afterwards, having a notoriously good memory for faces.

To portray the Hatter as a mad person may have served the story as a whole, especially considering the Cheshire Cat's remark that, '*We're all mad here*'. On the other hand, it may also have been a reference to the psychotic symptoms that hatters in 19th-Century England were prone to, due to the chemicals they used for preparing pelts. There is an ironic symmetry here with syphilis, the venereal disease known among the British as the French disease, which at the time was treated with the aid of mercurial ointments [17]. Having learned to soften pelts with the aid of mercuric nitrate from Huguenots originating from France, the British were equally quick to blame the French for the occupational disease that hatters developed after exposure to mercury. The technique to soften the outer stiff hairs of pelts, and make them pack together, involved dipping them into hot mercuric nitrate. Especially in poorly ventilated rooms, this easily led to mercurialism, a condition characterised by severe trembling and psychosis, not mentioned by the French Huguenots while letting their British colleagues in on this trick of the trade. Considering his interest in medical subjects, and his collection of medical books, Dodgson may well have known about the effects of mercury poisoning among hatters, or he may simply have borrowed the theme from common expressions such as '*the hatters' shakes*' and '*mad as a hatter*'. Thus, whether the Mad Hatter in the *Alice* story represents an actual person or not, there is reason to believe that Dodgson at least knowingly endowed him with contemporary notions about madness.

The same holds true for the Hatter's table companion, the March Hare. Whether he was meant to represent an actual person is unknown, but following Dodgson's request, Tenniel drew him with wisps of straw on his head, which at the time was a symbol, in art and on the stage, of being mad [18]. In *The Nursery Alice*, a later adaptation for children up to 5 years of

age, Dodgson accordingly informs his youthful audience, ‘*That’s the March Hare with the long ears, and straws mixed up with his hair. The straws showed he was mad - I don’t know why.*’ (Fig. 4.11) [19] Perhaps Dodgson really did not know why, and perhaps Torrey and Miller [20] are therefore right when they say that his descriptions of madness, such as those involving the Mad Tea-Party, were based on discussions with his uncle, Robert Wilfred Skeffington Lutwidge (1802–1873), a Lunacy Commission Inspector, whom he had accompanied at least once to an asylum.⁸

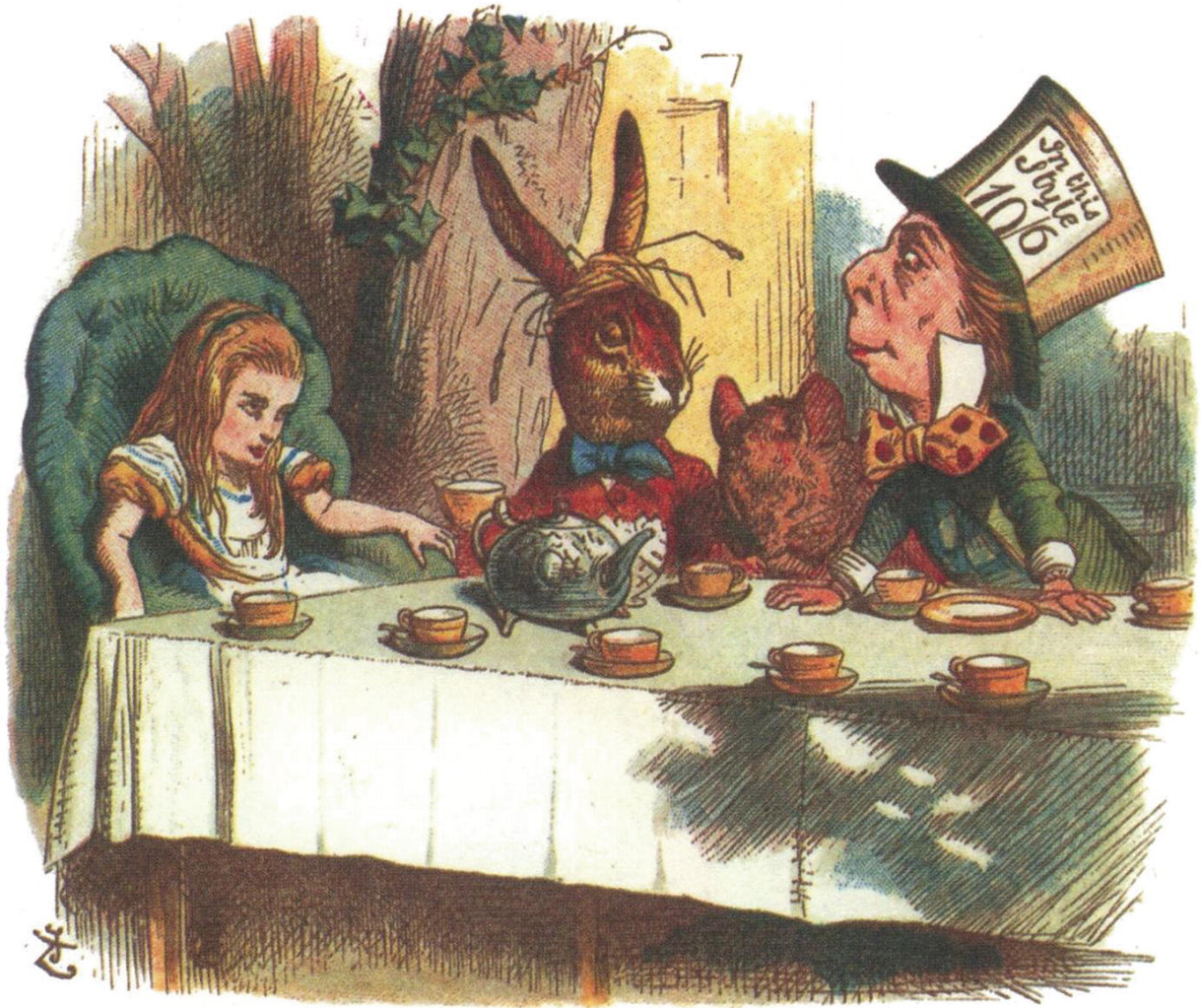


Fig. 4.11 *A Mad Tea-Party*, illustration by Sir John Tenniel (1890)

The Hatter’s other table companion, the Dormouse , might have been inspired by a wombat that Dodgson had seen lying asleep on a table at the house of his friend, the artist, Dante Gabriel Rossetti (1828–1882) [4, 22].

Another model may have been Thomas Jones Prout (1823–1909), Pro-Proctor at Christ Church, who had had a reputation for falling asleep at work, especially during long and boring meetings [23]. Falling asleep during tedious meetings is not uncommon in academic circles (or in fact anywhere), especially in warm and oxygen-deprived rooms. It may, however, be a sign of narcolepsy, a neurological disorder characterised by (among other things) excessive daytime sleepiness and falling asleep at inappropriate times and places. Whether Prout suffered from narcolepsy is unknown, but it has been suggested that Dodgson did intend the Dormouse to represent a sufferer from this condition [24, 25]. Alternatively, the sleepy little character may have been a parody on Dodgson himself [18], who during the trip on the Isis repeatedly pretended to fall asleep in front of Alice Liddell and her sisters.⁹

Other references to inhabitants of the academic bubble of 19th-Century Oxford include the Red Queen who, with her harsh demeanour and endless morals, appears to stand for the Liddell sisters' governess, Miss Prickett [26], while the Conger-Eel possibly stands for—again—John Ruskin (1819–1900) [27], England's leading art critic at the time, and a regular visitor to the Deanery, who taught Alice and her sisters drawing, sketching, and painting in oils—or, as the Mock Turtle says in the *Alice* story, '*Drawling, Stretching, and Fainting in Coils*'.¹⁰



Fig. 4.12 *Alice, the Duchess, and the Baby*, illustration by Sir John Tenniel (1890)



Fig. 4.13 *The Ugly Duchess*, oil on oak by Quentin Matsys (1513)

Thus, the *Alice* story introduces us to many thinly disguised individuals whom Dodgson and Alice Liddell had met in real life, represented in the story as people, animals, and mythological creatures. The same holds true for other story elements, such as the door to Wonderland, which appears to have been inspired by an actual door in the Liddell family's garden wall, and the treacle-well, which was inspired by an actual well at St. Margaret's Church, Binsey. Dodgson had once visited this well together with Alice Liddell, the water of which was said to be a healing cure for any kind of illness (hence the old English name treacle-well). Similarly, the great hall at

the beginning of the story is said to be inspired by the Great Hall of Christ Church with its rich history in real life and in cinema (having served, among other things, as the location for the Great Hall of Hogwarts in the *Harry Potter* films) [14].

Thus, even though the *Alice* story was marketed as a purely nonsensical book, it was riddled with story elements from a life that was all but very real. It was meant to be read for the mere fun of it, and anyone venturing to ask where all the nonsense came from was supposed to be placated by the final revelation that it all had been a dream —and yet, as things go in actual dreams, Dodgson had infused the story with elements from his waking life.

Needless to say, that does not imply that everything in the book was based on fact. How could it, in such an outrageously fantastical story? Apart from the pure fantasy elements, an important deviation from reality was that Dodgson presented Alice as a 7-year-old girl, whereas the real Alice had been 10 years old when he first told her the story. And then there is the issue of her likeness in Tenniel's drawings, since the Alice portrayed in the book does not look one bit like the Alice Liddell we know from photographs.

The reason why Tenniel drew Alice as a sweet little girl with bright blonde hair, rather than one with dark hair and a dreamy stare, was the outcome of another dispute with Dodgson. As Tenniel categorically refused to use models for his drawings, he initially saw no reason to make an exception for Dodgson. This first culminated in a bitter war over the question whether Alice should be drawn with a fringe or with 'bangs', such as those of Alice Liddell's, but probably upon realising that this would get him nowhere, Dodgson adopted an alternative strategy. Having spotted a photograph of a different girl, probably Mary Badcock, he decided that the drawings should be made in her likeness [4]. Even though the girl did not look at all like Alice Liddell (or perhaps because of that, if we wish to believe that he wanted to conceal too great an emotional investment in the Dean's daughter), he sought permission from her father to sit for Tenniel. Finally, he had to persuade Tenniel to break his habit and for once use a model. We do not know how he did it, but he succeeded and made Tenniel, grudgingly no doubt, undertake several trips to Ripon to sketch the girl.

Thus, however nonsensical the *Alice* story may seem, its constituent elements can often be traced back to persons, places, and events that had played a role in Charles Dodgson's and Alice Liddell's real lives.

Sometimes the connection was there for all to see, as with the parodies on popular verses, whereas other connections were more like in-jokes, meant for Alice and her sisters to enjoy in private. What the story behind Tenniel's blonde little Alice shows, is that there may also have been connections that Dodgson sought to conceal, like a dreamer who dreams up a substitute for something that would scare him out of his wits if he would encounter it in its original form. Whether he did have anything to hide has been the subject of much speculation, especially in connection with the pages—not to mention the several volumes—of the diaries that have mysteriously gone missing. But even without those parts, there is much more to know about him, including his relations with little girls, and the perceptual distortions that he had incorporated in the *Alice* book.

4.2 The Author of the *Alice* Book

If there is one thing that we know for sure about Charles Dodgson, it is that he was an inscrutable man. Even for a 19th-Century university tutor in mathematics, the kind of person who deserves to be pardoned for a quirk or two in advance, he had rather exotic tastes and habits, an idiosyncratic sense of humour, and clear-cut ideas about what he liked and disliked, to the extent that, in later life, he vowed never to attend 'invited' luncheons or dinner parties, since he did not fancy invitations. As a child, he had been exceptionally bright, being endowed with '*a very uncommon share of genius*', as his headmaster, Mr. Tate, had put it, and at Christ Church, he had gone on to excel and won various prizes, even though his mind had a tendency to wander to more frivolous things in life, and his achievements therefore often depended on brief outbursts of hard work rather than on steady labouring. After graduation, he had seamlessly slipped into the ranks of the university's established order and, remarkably for such a talented man, subjected himself to a rather dull, monotonous professional life, reluctantly teaching mathematics to dispirited young males, and working on mathematical problems and petty inventions at night, all the while staying in the same academic position. In the meantime, however, he gave his best time to the arts, writing stories, poems, and numerous letters, reading, seeing plays, visiting art exhibitions, and losing himself in what he called his '*one true passion*', photography. If university life was to Dodgson what the drinking of water is to the rest of us—of vital importance, but never

even remotely exciting—he had a habit of spicing up his life by indulging in the aesthetic pleasures provided by the arts.¹¹

As a corollary, the diary that he kept for over a period of 55 years¹² (which, as we saw, has not survived in its entirety) constitutes as much a collection of mini-reviews of books, plays, poems, sketches, drawings, paintings, photographs, exhibitions, landscapes, and travel destinations, as it is an account of his personal experiences. Stylistically, it has much in common with 21st-Century Twitter accounts, comprising brief bursts of text messaging, saying, ‘*Been there*’, ‘*Seen that*’, ‘*Met X*’, ‘*Photographed Y*’, ‘*Got an autograph from Z*’, and so on.¹³ Obviously, there was no one at the receiving end of this Twitter account *avant la lettre*, but Dodgson kept updating it obsessively, sometimes summarizing several months on a single page, and at other times faithfully chronicling exciting developments in day-to-day accounts.

Thanks to the diary, the numerous letters (estimated to run over 100,000, of which a modest 10% have been published), and the testimonies of people from his extensive network, a relatively large amount is known about Dodgson—so much in fact, that he has become the kind of public figure that is easily confused with a close acquaintance. After all, how many people do we know so well that we are familiar with their eating and sleeping habits, the way they prepare their tea, the books and poems that they like and dislike, the goals they set for themselves in life, the way they think about God, religion, and fairies, and the exact type of boots they like children to wear?

From this wealth of written material, Dodgson emerges as a slender Englishman, 5 feet and 10 inches tall; thin, with a pale complexion, and deep, blue or blue-grey eyes, set in a boyish—some have said ‘*feminine*’—face that was slightly asymmetrical, with the left eyelid drooping a little, and the corners of the mouth at different heights, responsible for lending him a ‘crooked smile’. His hair used to be rather long for the fashion of the day, as if it always needed cutting. He was stiff in gait as well as in manners, preferably clad in the clerical dress of the day, including a black frock coat and a white cravat, and wearing a top hat and black-and-grey woollen gloves¹⁴ in all seasons, although never an overcoat if not absolutely necessary. Although he did look frail, his handshake was said to be firm and strong. When he walked, he was so erect that he almost seemed to keel over backwards, and his gait has been described as ‘*queer*’ and ‘*jerky*’, as if he

suffered from a defect known as ‘housemaid’s knee’ [4], an observation most likely made during one of his recurring episodes of ‘synovitis ’ (i.e. inflammation of the knee). The same may be true for the observation that his left shoulder appeared to be a little higher than the right one. Living an almost ascetic life, he used to eat modest quantities and for many years, whenever he had the chance, took daily walks around Oxford, thus keeping his physical condition in proper shape and his head clear of the daily hassles. His preferred alcoholic drink was sherry (with a biscuit). He prepared his tea by walking up and down the room, teapot in hand, for exactly 10 minutes, claiming that that would give the best results. Sleeping was a luxury not always granted to him, as he often used his nightly hours to prepare his work for the next day, hating to show up unprepared for his lectures or let his students down when he had promised to correct their assignments. In addition, he used those hours for reading and writing, as well as for designing gadgets that were supposed to make life easier, such as a wallet for stamps (called the *Wonderland Stamp Case*) and a device he called the *Nyctograph*, which allowed him to take notes in the dark. Perhaps he also suffered from insomnia due to recurring worries of whatever nature, but that is something we cannot be sure about. He hated ballet, he hated flowers, he hated men dressed as women on the stage, he hated crimson (although not pink), he hated babies, he hated high-heeled boots with pointed toes, he hated vulgarities, he hated untidiness, and, most of all, he hated it when others associated him in public with the book that had brought him international fame, condemning those who dared to single him out in front of an audience as the author of *Alice’s Adventures in Wonderland*.¹⁵ He spoke in a high-pitched voice and was often shy among adults, stammering slightly in their presence, but enough so to make him dread performing in public. In his capacity as a tutor, and as a Deacon for the Church of England, this was obviously a serious obstacle. And yet he never seemed to shun from his professional responsibilities once he had agreed to fulfil them, delivering his lectures and sermons as well as he could manage.

As recounted by many of those who knew him, his stammer instantly melted away in the presence of children, who somehow had the effect of turning him into an eloquent, sparkling, humorous person on the spot, free from awkwardness, and unafraid of making a fool of himself, sitting under tables with his child friends to participate in their games and, according to

Reed [2], even crawling into a house once on all fours, growling like a bear, only to shock the owners and leave them behind dumbstruck as soon as he had found out that he had picked the wrong address. Whether Reed's story is based in fact is not entirely clear, but the way he behaved in the company of children makes it possible that something like this may indeed have happened.

In his contacts with children, Dodgson was a uniquely gifted person who knew how to grab their attention with a single word or gesture, and who communicated with them as if they were his equals, even though he never seemed to lose sight of his own position as an adult, and never shed the role of teacher that suited him so well. Although his interest in them may have had a self-serving aspect, many former friends have testified that they had felt understood by him, and that he had played a unique role in setting them on their path to whatever it was that they wished to pursue. His interest in children was natural and genuine, although perhaps not entirely universal. He liked to say that he was very fond of girls who had not yet reached the age of puberty, especially when they displayed the kind of spontaneity and openness that allowed them to blossom in his presence, just as he could blossom in theirs. Additional points were given for fair hair and for going by the name of Alice. That fondness appears to have been balanced, for a while at least, by a deep-felt contempt for those who had gone on to develop secondary sexual characteristics. As he growled in his diary on behalf of a girl named Lucy Tate (1842–1873), for example,

Lucy Tate has grown from romping girl into the most staid of young ladies, so much change can 2 years produce [29].

Even Alice Liddell was not spared from this type of comment. As he wrote of his favourite child friend after she had just turned 12,

Met Alice and Miss Prickett in the quadrangle. Alice seems changed a good deal, and hardly for the better, probably going through the usual awkward stage of transition [30].

According to some diary entries, boys could hardly do any better with him. Irrespective of their age, Dodgson considered them a '*tribe of young roughs*' and, with the exception of a few—such as Alice's brother, Harry Liddell—he claimed to have no interest in them whatsoever. However, later

entries show that he did take an interest in them after all, and that he successfully built friendships with a good many of them. Gradually, his dislike for girls above a certain age—if it had ever truly existed—also melted away, as in later years, he sometimes wrote appreciatively about adolescent girls whom he had met. Moreover, as his child friends grew older, he often maintained a keen interest in them and stayed in touch with them long after they had reached adulthood, including, of course, Alice Hargreaves *née* Liddell, although Alice herself appears to have kept him at a distance for most of her life. That said, little girls rather than photography appear to have been his ‘*one true passion*’, and until the end of his life, he could not resist scouting potential child friends, approaching them unashamedly in streets, in theatres, in schools, in playgrounds, and on beaches, jotting down their names in his diary with the obsessive precision of a collector, meanwhile building a tangible collection of images of little girls as represented in paintings, drawings, sketches, photographs, and sculptures, to which he was always drawn as if by a magnet. One glance at the long list of people he counted as his friends is enough to convince us that, throughout his life, Dodgson had built himself a network that would make many of today’s Facebook adepts turn green with jealousy. It is also obvious from that list, that people of both sexes, and of all ages, were amply represented, including scores of adult women, some of whom he used to visit privately and unchaperoned.¹⁶ Therefore, despite his speech impediment and social awkwardness, it appears that he was remarkably capable of building and managing a gigantic network of friends and acquaintances. It was just that he enjoyed the sight and company of prepubescent girls over anybody else’s—or so he would have everyone believe.

Thus, for a man who once wrote, ‘*My constant aim is to remain, personally, unknown to the world*’, there is a lot that we do know about Dodgson. This also begs the question of why he kept a diary in the first place and for whom he believed to be writing it. Unlike today’s Twitter users, Dodgson cannot have been aware of the scope of his potential audience. Nevertheless, it is clear from the way he wrote that he kept his diary with the intention of preserving information for posterity, making sure to list all and any personal achievements, from a raise in his salary, and the acceptance of a manuscript, to the beginning of a new poem or even the title for a new work, even if that work stood little chance of coming to fruition.

He also appeared to consider himself an expert on virtually anything, chronicling how he had been dealing out advice, solicited or not, on architecture, timekeeping, ciphers, stage plays, lawn tennis tournaments, prostitution, child labour, lunar observations, works of art, photography, the postal system, money orders, railway tickets, vivisection, censorship, voting rules for elections, horse betting, child's clothing, hydrophobia¹⁷ and other medical issues, pedagogic issues, dinner etiquette, women's rights, and the fate of the people of Tristan da Cunha.¹⁸ It is this meticulous chronicling of all his talents and achievements¹⁹—although interspersed with guilty lamentations about nondescript shortcomings that he apparently detected in himself—that makes one wonder whether Dodgson knew, or even hoped, perhaps, that his diaries would be read by others. The fact that he at least considered it a possibility is nowhere more apparent than in a passage where he writes about the mother of a child friend in the theatre, and says, *'I do not mention her real name, as she does not wish it known, her relations not knowing that she is on the stage'* [33]. So, we may ask ourselves, why all the cloak and dagger, if there were no intended audience for the diaries? Perhaps anyone who keeps a diary is aware, to some extent at least, that what is trusted to paper may be read by others—and yet the way Dodgson wrote, was almost as if he wanted his diaries to find their way to the greater public.

Thus, although we do know rather a lot about the man, there is at least as much that we do not know about him. One way of looking at anyone's diaries is to see what is written in them. Another way of looking at them, is to see what was left out—and Charles Dodgson left out a lot. Here I am not speaking of the trivialities that make up the bulk of our ordinary lives, but chunks of vital information on crucial yet underexposed aspects of his life.

For starters, one thing that Dodgson never wrote about in his diaries was his membership of the *Society for Psychical Research* (SPR). Founded in 1882 and headed at the time by the Cambridge moral philosopher Henry Sidgwick (1838–1900; Fig. 4.14), the SPR saw it as their task to scientifically investigate the paranormal. After Charles Darwin (1809–1882; Fig. 4.15)—with whom Dodgson had corresponded—had introduced the greater public to the notion that Man had descended from primates rather than being created by God, and people had subsequently turned to science to find answers that had traditionally been provided by the clergy, there suddenly was a whole new area for scientists to explore. Not only was

Dodgson a member of the SPR, he was one of its founding members and, moreover, remained involved with the organisation until his death [34]. As mentioned in some of his letters (although not in his diaries), he used to read books on spiritualism , and from entries scattered throughout his diaries, we also know that he held a lifelong fascination with ghost stories. In one such entry, he records how he,

...met Mrs. Wall, from India, who told me strange tales of Indian magic , pigeons put into bottles, thread drawn out of any part of the chest or arm of the performer, and a fulfilled curse on three men, that all should die violent deaths in six years [35].

Sometimes he even seemed to believe that he himself might have had a psychical experience of sorts. As he wrote, for example,

At the evening service of Christ Church , a curious thing happened, suggestive of ‘telepathy .’ Before giving out the second hymn, the curate read out some notices. Meanwhile I took my hymn-book, and said to myself (I have no idea why) ‘it will be Hymn 416,’ and I turned to it. It was not one I recognised as having ever heard: and, on looking at it, I saw ‘it is very prosaic: it is a very unlikely one.’ And it was really startling, the next minute, to hear the curate announce ‘Hymn 416!’ [36]

Likewise, all the other founding members of the SPR had been intrigued by phenomena such as clairvoyance , deathbed visions , Poltergeists , telepathy, telekinesis , mesmerism , animal magnetism, and glimpses of a life after death, and wished to investigate them scientifically. Doing so under the supervision of the austere Sidgwick implied that they aimed for nothing less than solid scientific proof—or refutation, as they were all well aware that the field was riddled with superstition, trickery, and fraud. Riding on the wave of occultism that washed over the recently secularised West, the SPR set out to scrutinise the work of psychical mediums , attending séances in which spirits were summoned, tables rose and fell, decent ladies spawned cobwebs of ectoplasmic substance from their mouths, and heavy objects were seen miraculously flying through the air. There were card-guessing experiments, experimentations with the telepathic induction of trance states , mind-reading tests , studies of automatic writing

, crystal-gaze experiments, and much, much more—the one study being even more exotic than the other.²⁰

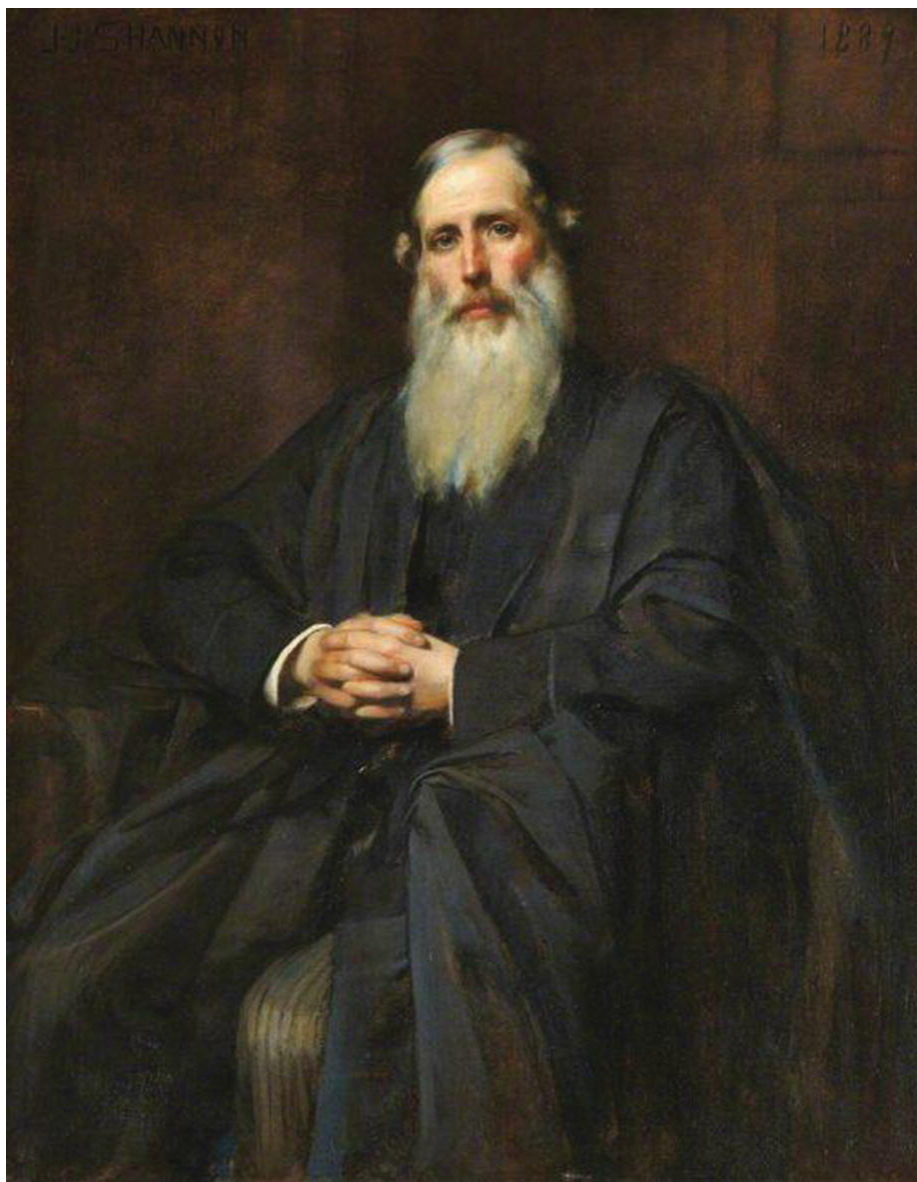


Fig. 4.14 Henry Sidgwick, *Knightbridge Professor of Philosophy*, oil on canvas, by Sir James Jebusa Shannon (1889)

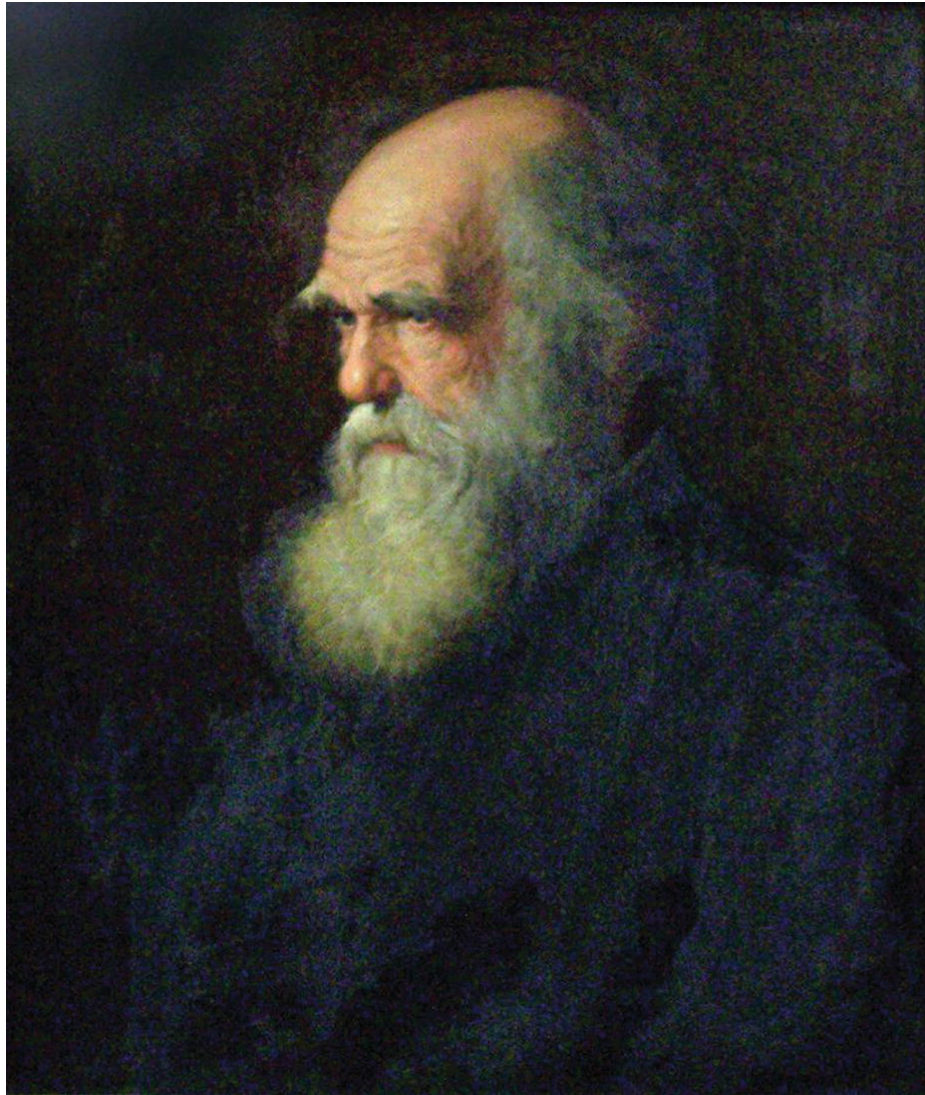


Fig. 4.15 *Charles Darwin*, oil on canvas, by Walter William Ouless (1875)

As a charter member of the SPR, and being much interested in miracles²¹, ghost stories, and other supernatural themes, Dodgson must have followed those studies closely and been impressed—or at least baffled—by their results, as were many of his fellow academics at the time. Establishing once and for all the validity of metaphysical claims, rendered obsolete by the scientific revolution, by means of science's own methods, no less, was undoubtedly the most exciting project that any man of learning could have dreamed of at the time. It was no wonder, therefore, that the SPR counted among its members so many prominent scientists and dignitaries.²² It was probably the most prestigious, and—as we would now say—*coolest* organisation that any academic could be part of near the end of the 19th

Century. One would have expected Dodgson to at least mention his membership of this illustrious Society in his diary, if only to express the honour bestowed upon him by moving in these distinguished circles. However, he is completely silent in this respect, not reflecting a single time on his membership or on the consequences of the SPR's spectacular findings.

The same holds true for his involvement with the *Ghost Society for Paranormal Investigation* and the Freemasonry . Although from time to time he indicated in his diaries that he had attended fetes or other gatherings at Freemason's Hall, he never mentioned whether (or not) he was a member, whether he had read any books on the subject (even though he owned several) or whether he believed in the principles of this gnostic tradition. Likewise, it is known that he was interested in Theosophy and Rosicrucianism , two other gnostic traditions about which he never wrote in his diaries.²³

Another topic that he hardly wrote about was conflict. Although aggressive thoughts generally make us feel uneasy, everyone runs into a conflict now and then, and a diary seems to be a safe place to ventilate one's frustrations over unresolved issues. We know that Dodgson was relatively quick to disagree and sometimes hardly knew how to hold back while criticising an opponent; however, in his diaries, he suggests that a falling-out rarely happened in his life and that, if it happened, he was a master at resolving this without giving away his emotions.²⁴ Nevertheless, sources around him indicate that he lost his temper quite quickly, and that conflicts were much more common than suggested by him. By comparing his travel diary of 1867, for example, with that of his friend, Henry Liddon—who had accompanied him on his trip to Russia—we find that the latter records various '*great arguments*' with Dodgson on religion and other topics, whereas Dodgson in his entries for those same days quietly reflects on trivia. In another entry, Dodgson matter-of-factly remarks that he would rather not attempt to describe the Cathedral of Cologne any further than by saying that it was the most beautiful of all churches he had ever seen, or could imagine [42], whereas Liddon notes that,

Dodgson was overcome by the beauty of the Cologne Cathedral. I found him leaning against the rails of the Choir and sobbing like a child. When the verger came to show us over the chapels behind the

Choir, he got out of the way: he said that he could not bear the harsh voice of the man in the presence of so much beauty [43].

Emotion in general, then, was a topic that Dodgson hardly wrote about. A play or a piece of art could be '*the most joyous thing*' he had ever seen, but which particular emotions it evoked was something that he did not share, at least not in his diaries. This was also noted by the Carrollian scholar Edward Wakeling , who says,

[Dodgson's] private journal was principally a way of noting events in his life, people he met, and places he visited; it was not a vehicle for expressing his inner-most thoughts and ideas. Surviving journals are, to a large extent, matter-of-fact and dispassionate [23].

As a corollary, perhaps, another pair of themes conspicuously absent from Dodgson's diaries is romantic and sexual desire . Having remained a bachelor for all his life, there is one single passage (on financial matters, no less) where he ponders setting aside some money in case he might ever wish to get married [44]. It is the only reflection on offer in his diaries on the possibility of a marriage. As far as we know, a love interest, female or male, was never on the cards, even though, throughout his life, Dodgson met numerous potential candidates for marriage and built long-lasting friendships with many of them. He also had an eye for physical beauty when it came to photography and art and frequently indicated in his diaries whether he liked someone as a conversational partner or not. And yet nowhere does he comment on anyone's sexual or romantic attractiveness, even though the girls and women with whom he had surrounded himself were often so beautiful, that one of his biographers sighed that he must have been '*addicted to physical beauty*' [31]. Only his first biographer, Stuart Dodgson Collingwood , suggests, in a single sentence, that Dodgson may once have been rejected by a woman whom he loved, even though the rest of his family have always stressed that he had no interest in women whatsoever [16]. Consequently, it has traditionally been assumed that he had no sex life either, although it would seem more accurate to say that on the basis of these remarks he would not have seemed to have had a sex life *with a partner*. Whether that justifies the conclusion that he had been asexual in nature is hard to tell, even though it was suggested by

Collingwood , and has since resonated with Dodgson's readership, who even during his life came to perceive him as an almost sacrosanct, mythical figure, living on the fringes of mainstream society and being devoid of the more visceral aspects that dominate ordinary lives.

Obviously, themes such as sexual orientation and behaviour were not openly discussed in Victorian England. At the time, even scientific research in these areas was still very much in its infancy, not to say sidelined and neglected. And yet asexuality in adults was something that had already been described by the medical profession. The great pioneering work, *Psychopathia Sexualis* , by the German psychiatrist Richard von Krafft-Ebing (1840–1902), already contained references to *anaesthesia sexualis* , as he called the absence of sexual instinct [45]. His book was widely read and translated faster (and initially into more languages) than *Alice's Adventures in Wonderland*, so we can safely assume that it quenched a certain thirst for knowledge among contemporary psychiatrists (who, incidentally, did not comprise the exclusive readership of this medical bestseller). Even though it took until the 1940s before human sexuality went on to become a mainstream scientific theme, it took much longer for asexuality to be explored. In fact, it has only fairly recently become of emerging interest, perhaps because it is traditionally considered to be exceptionally rare. As indicated by a recent study carried out in New Zealand, among 18,261 adults, only 0.4% of all women and not even 0.1% of all men consider themselves asexual [46].

Whether Dodgson did have any sexual drive —let alone a sex life, which he then must have hidden well from others—is a matter of pure speculation. Even more speculative is whether the myriad lamentations and indications of self-loathing throughout the first few volumes of his diaries may have had anything to do with sexuality . Those passages always seem to appear out of the blue, lacking any context and bearing no apparent relation to anything that went before. As the integral transcription of the diaries demonstrates (contrary to the edited version of 1953 [47, 48], in which the majority of those outcries had been suppressed), there really were a lot of them. A few examples will suffice to get a general idea:

Make me a clean heart, oh God , and renew a right spirit within me [49].

Help me for Christ's sake. Amen. I write this in my photographic studio, with the earnest hope that from this may date, by God's blessing, the commencement of a new and better life. The spirit indeed is willing but the flesh is weak. Help me, for Christ's sake. Amen, Amen [50].

I pray to Thee, oh God, for thy dear Son's sake, help me to live to Thee. Help me to overcome temptation: help me to live as in Thy sight: help me to remember the coming of death. For myself I am utterly weak, and vile, and selfish. Lord, I believe that Thou canst do all things: oh deliver me from the chains of sin. For Christ's sake. Amen [51].

For a man who appears to have lived nothing but a decent life, it is hard to imagine what the reason for these prayers and outcries may have been, other than, perhaps, his own scrupulous conscience. It should not surprise us, therefore, that there has been some doubt as to whether Dodgson really lived such a decent life, and that people started speculating about the relations with his child friends, notably in connection with his photographs, some of which notoriously show them in the nude. These speculations are not entirely ungrounded, as the first time he recorded having made such photographs his diary entry makes for an unfortunate combination of themes:

Mrs. Latham brought Beatrice, and I took photographs of the two; and several of Beatrice alone, sans habilement. Read Lowe's great speech of last night against 'household suffrage.' Began reading the 'Epistle to the Galatians' in Greek. I have much neglected one means of grace, the reading of the Bible, and desire, with God's help, to begin a better course. Help me, oh Lord, for Thy dear Son's sake, to turn to Thee in true penitence for my sins, and lead me in the right way! For Jesus' sake. Amen [52].

Here, for once, we find the themes of nude photography and the need for repentance in relatively close proximity to each other in a single diary entry.²⁵ As in any other field that is so radically polarised, it is somewhat of a challenge to acknowledge that we simply do not know what we are dealing with here. For an adult to have no sexual interest whatsoever is rare.

Whether Dodgson did (or not) is impossible to tell on the basis of what is currently known about him. As regards his feelings for little girls, however, I believe that Florence Becker Lennon was right when she challenged any sensationalist interpretations, and instead pointed out the overwhelming number of testimonies of former child friends who reported nothing but the fondest of memories of him:

He was his own best chaperon, and while he did a good deal of kissing, he seems to have known, with his usual delicacy and respect for others, when to stop. His little girl friends would not have remembered him with such unanimous glee if he had not always been a good and trustworthy friend to them. There is total lack of embarrassment in all these dozens of memoirs, which, even after all these years, rings true [4].

Victorian England was unfamiliar with phenomena such as *#MeToo*, but one would have expected at least some testimonies of sexually transgressive behaviour or romantic involvement to have surfaced over the years, if Dodgson had ever crossed that line.²⁶ I will return later to this enigmatic aspect of Dodgson's life, but let us first take a look at yet another theme that is so little exposed in his diaries, that is, his health. What interests us here, of course, is exactly that. It is the focus of the present chapter, and the only proper avenue to finding out whether Dodgson himself had experienced the remarkable perceptual phenomena that currently make up the Alice in Wonderland syndrome.

4.3 Dodgson's Health: A Reconstruction

At the age of 57, Charles Dodgson boasted that he had '*never known serious illness*'. At 64, a year before his death, he wrote a letter to his sister Louisa, stating likewise, '*My health is something to be most thankful for.*' Scattered throughout his correspondence, we find similar statements, indicating that this was his standard way of communicating about his health. Sometime before his letter to Louisa, however, he had apparently been less content, as he had followed the advice of his physician to purchase a *Whitely Exerciser*, a chest expander operating by a complex series of rubber strands and pulleys that was designed to build the muscles

(Fig. 4.16) [53]. As he wrote to her in the same letter, the device had helped him to regain much of his former strength and had allowed him to make 18-mile walks again, several times a week. That was not as far as he had done in the past, when he did 23 or even 27 miles at a brisk pace; nevertheless, this still indicates that he was in admirable physical shape at the time—which, indeed, he had been for most of his life.



The advertisement features a black and white illustration of a family of four using 'The Whately Exerciser'. A man and a woman are standing and holding onto the device's frame, which is suspended by ropes. A young girl is sitting on the device's base, and a small child is standing next to her. The device is positioned next to a Christmas tree. The text 'SANTA CLAUS IS COMING' is at the top, and 'The Most Sensible Christmas Present' is on the left. Below the illustration, it says 'FOR EVERY MEMBER OF THE FAMILY' and 'THE WHATELY EXERCISER'. A paragraph of text describes the device's benefits, and the company name 'WHATELY EXERCISER COMPANY' is at the bottom, followed by three addresses: 'Room 41—203 Fulton Street, New York.', '121 Randolph Street, Chicago, Ill.', and '821 Mutual Life Building, Buffalo, New York.'

SANTA CLAUS IS COMING

The Most Sensible Christmas Present

FOR EVERY MEMBER OF THE FAMILY

THE WHATELY EXERCISER

You cannot give a better Christmas present—suitable for any home, a constant pleasant reminder of the giver. It is neat, convenient and durable. Provides indoor exercise for office men, women, students, boys and girls. Its use brings every muscle into play; makes sluggish blood active, developing muscle and quickening the intellect; makes weak women strong, giving them good form, good complexion, good health; aids growing children. Write the nearest address below to-day for **Free Illustrated Book** and special holiday price.

WHATELY EXERCISER COMPANY

Room 41—203 Fulton Street, New York.
121 Randolph Street, Chicago, Ill.
821 Mutual Life Building, Buffalo, New York.

Fig. 4.16 Advertisement for the *Whately Exerciser* (c. 1905)

With a height of around 1.78 m and a weight of 65 kg, measured at the age of 50, he had had a body mass index (BMI) of 20.5. As he has always been described as thin, this means that he must have been in the lower range of a healthy weight for most of his life, although he may occasionally have dipped below the line. Key ingredients to his health were his habit of not smoking, his almost daily exercises (walking and, later in life, riding his *Velociman* , ²⁷ pulling at his *Whitely Exerciser*, and practising with dumbbells), eating simple, small meals, and preferably skipping midday meals or contenting himself with a glass of sherry and a biscuit (or a slice of melon with ginger or sugar) as he claimed to have no appetite around lunchtime anyway. How often he drank sherry on an empty stomach is unknown, but if his overall preoccupation with a healthy lifestyle is something to go by, this can hardly have been a daily habit. That he used a *Ferrometer* , a device to purify water, indicates that he was very conscious of the potential threat of cholera and other diseases lurking in domestic drinking water [55]. Seeing his dentist very frequently meant that he also managed to stay in possession of his teeth until the end of his life, which was an accomplishment in itself at the time.²⁸

When he died in 1898, at the age of 65, it was probably due to pneumonia, following a sickbed that had started out as ‘*a feverish cold of the bronchial type*’ (or so his physician, Dr. George Dabbs (1845–1913), had called it). A mere 10 days before, Dodgson had still been in the full swing of his life, writing, inventing new mathematical rules, preparing texts for publication (one of which appeared in the top scientific journal *Nature*), walking, exercising, and seeing ‘child friends’ old and new, so nothing had indicated that the end had been quite so near. Moreover, having suffered from bronchitis numerous times before, Dodgson had probably been the last to expect this fatal turn.

Or perhaps he had seen it coming after all, as 65 years was a respectable age at the time. Moreover, 7 years before, while suffering from a sustained headache, he had written in a letter that he felt ‘*rather ill*’, adding,

I don’t think I have any right to reckon much on the coming years, and there is a great lot of work I want to finish before the end comes [48].

That Dodgson had such gloomy thoughts at the relatively young age of 58, probably had something to do with the way he had felt during his sickbed (and indeed many times before, as this was not his first reflection on his own mortality). However, for a 19th-Century Englishman such thoughts were also realistic. Men in the UK, born around 1830, had a life expectancy at birth (LEB) of 40 years. The reason why the LEB was so low, was primarily because of the high number of infant deaths due to infectious diseases. As a consequence, anyone who made it past childhood was automatically rewarded with a much higher position on the life-expectancy charts, although 65 remained an age to be grateful for, especially if one succeeded to stay in as good a shape as Dodgson.²⁹ And yet, despite all the boasting and thankfulness, an overview of his medical history indicates that there had been plenty of ailments for him to deal with in life. Perhaps it is not entirely fair to say that he hardly reflected on this in his diaries but, as we shall see, his entries on health issues were in the same ‘Twitter’ style as all others, even though he was much concerned with his health and had a habit of consulting top specialists to secure the best medical advice he could get.

Since there are no medical records to complement Dodgson’s own brief entries, it is not always easy to fully appreciate what he had actually suffered from. Nevertheless, in what follows, I attempt to reconstruct Dodgson’s medical history as faithfully as possible. Since any attempt at completeness would be ill-fated, I thought the least I could do was to display all that is known about his health, including minor conditions and injuries, which nonetheless need to be taken into account if we are to arrive at a carefully weighted opinion on whether or not he had experienced perceptual distortions himself and, if so, what might have been the underlying cause. The last thing that I would want here is to go out on a limb. That said, if you are not that much interested in the particulars of Dodgson’s health, you can always skip this part and either take a glance at the overview in Table A.1 (Appendix A), or else fast-forward to Chap. 7, where I provide a summary of Dodgson’s medical history, and go on to explain what we can and cannot conclude from it.

4.3.1 Hearing Problem

Dodgson liked to boast about his health. And yet his suggestion that there had been nothing to complain about can perhaps be best taken as an

indication that he was no cry baby, even though, healthwise, it had not all been smooth sailing for him . For one thing, he had been hard of hearing in the right ear from early childhood onwards. Exactly at what age that came about is unknown, but while at Rugby School, he had attracted a febrile condition, most likely infectious in nature, and possibly complicated by otitis media, that had left him with a hearing problem. Then, at the age of 17, he attracted mumps , an infection caused by an RNA virus of the *paramyxovirus* group, which usually presents with fever , tiredness, muscle pain, headache, and swelling of the parotid glands. Mumps can be prevented by the mumps vaccine, usually administered in two doses—however, in Dodgson’s time, the cause was not yet known and the vaccine had not yet been developed. Consequently, the disease was much more prevalent, as were its complications.

Mumps can be complicated by meningitis (an inflammation of the membranes enveloping the brain), pancreatitis (an inflammation of the pancreas), orchitis (testicular inflammation), and hearing impairment . Whether Dodgson went on to develop any of the other complications is unknown, but he did become permanently deaf in the right ear, with the mumps as the most likely cause. As his mother, Frances Jane Dodgson-Lutwidge (1803–1851), wrote to her sister Lucy at the time,

In [Charles’s] letter received on Tuesday he said that the mumps had gone but that they had left him much more deaf than usual - this we trust is quite to be accounted for from the nature of the complaint and may probably last longer than the visible swelling of the glands [57].

Contrary to what Mrs. Dodgson had hoped, the deafness in the right ear would stay with him for the remainder of his life. We now know that deafness, even of the unilateral kind, is a risk factor for tinnitus (ringing in the ears), verbal auditory hallucinations (hearing voices), and musical hallucinations (hearing music out loud in the absence of an external source) but nowhere in his diaries do we find any indication that Dodgson suffered from such perceptual complications. Nonetheless, his hearing problem caused him considerable trouble, especially in his professional life, in social conversations, and in his appreciation of music. To optimise hearing with his unaffected ear, he preferred people to walk on his left. In theatres and

concert halls, he preferably sat at the extreme right. Whether he took any other measures is unknown, but what we do know, is that he kept worrying about his hearing, and kept hoping that something could be done about it. At the age of 24, he even called on Dr. Joseph Toynbee (1815–1866), one of the greatest aural surgeons of the 19th Century, to seek his advice [58]. What Toynbee told him has not been handed down but, as so often with deafness, he was unable to do anything about it.

4.3.2 Face Blindness

Another common thread running through Dodgson's life was a bad memory for faces. This was a peculiar and oddly specific handicap, which caused him much embarrassment in social situations. Especially since his memory for all else in life was nothing short of remarkable, allowing him to do elaborate sums in his head, and reproduce dates and other numbers with the aid of advanced memory techniques (the *Memoria Technica* of his own devising), it must have been a kind of disgrace for him that other people's faces failed to register sufficiently to make any lasting impression on him. His attempts to deal with this social handicap—which it was, for lack of a better term—led him to employ unusual strategies, including attempts to memorise the faces of acquaintances from photographs. As he wrote to Lily Falle (1859–1957), for example,

...I petitioned for a photograph of you. Really, it would be a most useful thing to have: my memory for faces is wretchedly bad: and yours is, I fear, beginning to fade out of it: and it is quite possible that if we were to meet unexpectedly, say in Regent Street, I might not recognise you! [32]

There is a neurological condition called prosopagnosia , or face blindness , which produces this symptom. It is not a literal blindness for faces, in the sense that one is unable to perceive faces at all, but rather an inability, or strongly diminished ability, to memorise, recall, and/or recognise faces. People suffering from prosopagnosia typically lack the ability to perceive differences between individual faces and may need to rely instead on striking accessories such as a moustache, glasses or an unusual hair style, to recognise a person. In *Through the Looking-Glass* , the stylistically watered-down sequel to *Alice's Adventures in Wonderland*, Dodgson

presents the egg-shaped Humpty Dumpty (Fig. 4.17) as a creature afflicted with this condition, while having him dish out a remarkable piece of advice to Alice when she is about to leave:

‘Is that all?’ Alice timidly asked.

‘That’s all,’ said Humpty Dumpty. ‘Good bye.’

This was rather sudden, Alice thought: but, after such a very strong hint that she ought to be going, she felt that it would hardly be civil to stay. So she got up, and held out her hand. ‘Good-bye, till we meet again!’ she said as cheerfully as she could.

‘I shouldn’t know you again if we did meet,’ Humpty Dumpty replied in a discontented tone, giving her one of his fingers to shake: ‘you’re so exactly like other people.’

‘The face is what one goes by, generally,’ Alice remarked in a thoughtful tone.

‘That’s just what I complain of,’ said Humpty Dumpty. ‘Your face is the same as everybody has - the two eyes, so----’ (marking their places in the air with his thumb) ‘nose in the middle, mouth under. It’s always the same. Now if you had the two eyes on the same side of the nose, for instance - or the mouth at the top - that would be some help.’

‘It wouldn’t look nice,’ Alice objected. But Humpty Dumpty only shut his eyes, and said ‘Wait till you’ve tried.’ [59]

With his outrageous piece of advice, Humpty Dumpty obviously asks for the impossible. However, what he does convey with remarkable accuracy is the level of confusion that people suffering from prosopagnosia may experience. To be able to tell one person from another, they usually have to rely on truly striking facial features. Alternatively, they may recognise people by their voice, their clothing or certain manners. Oliver Sacks famously suffered from this condition, which made it hard for him to react spontaneously to acquaintances whom he ran into unexpectedly. Dodgson

appears to have had the same problem, judging by what he wrote in a letter to Arthur Gilkes (1849–1922), whose presence he had apparently overlooked in the Common Room of Christ Church :

Now let me take the opportunity of offering the most ample apology, that words can convey, for my stupidity in not recognising you when you called... I had not forgotten meeting you in Common Room the night before: but I have so bad a memory for faces that I have very small confidence in the look of anybody, as evidence of who he is. I had not the smallest expectation of seeing you, and I was expecting to see a Mr. Humphery (whom I do not know by sight)... So, though I did notice the extraordinary resemblance of the unknown Mr. Humphery to the known Mr. Gilkes (I believe some such idea as 'how bewilderingly alike people are!' crossed my mind), I did my best to see in him the man I was expecting. Believe me that I am much ashamed for having failed to recognise you, and hope I shall not be so stupid again! [60]

We all have varying talents to memorise faces, and we may all fail to recognise a familiar face from time to time (due to an ordinary 'slip of the mind' called *underidentification*), but the frequency with which Dodgson mentioned this problem is indeed remarkable. It is therefore tempting to speculate that his prosopagnosia may also have been an important reason why he was so adamant on orchestrating his meetings with other people, to the extent that he often urged people to visit him alone, or at least not to bring strangers. It is unclear at what age he developed this condition, or whether it had perhaps been present since birth. We now know that the fusiform gyrus , a bilateral structure at the base of the brain, plays an important role in the recognition and representation of faces and that structural (and perhaps also functional) damage to that area is a likely cause of prosopagnosia. Whether Dodgson suffered from this type of brain damage is unknown, and it is impossible to establish this, even with the benefit of hindsight. However, on the basis of his own reports, this may well have been the case.

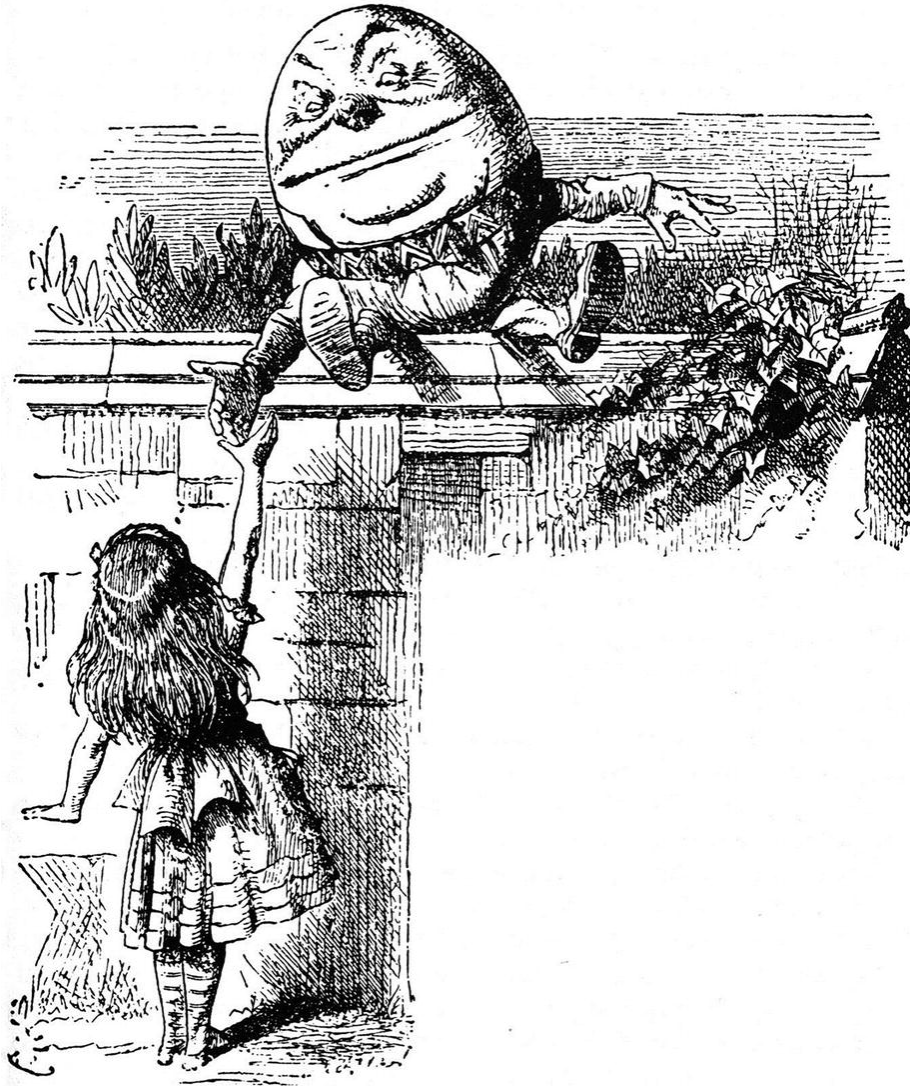


Fig. 4.17 Alice Meeting Humpty Dumpty, illustration by Sir John Tenniel (1871)

4.3.3 Speech Impediment

Then there was the speech impediment, of which we do know that it had been present from early childhood onwards. Characterised by some biographers as a stammer, others maintain that Dodgson had difficulty with adjacent words beginning with the same letter, and still others that he had difficulty with the letter *p*, and therefore spoke in a hesitating sort of way, slowly and precisely, avoiding the *p*'s, and taking his time searching for alternative expressions. Such a hesitation or pausing before speech is called 'blocking', although technically it may be a consciously employed trick to buy oneself some time for word substitution. Whatever its true nature, it

caused Dodgson considerable trouble. As recalled by his former child friend, Isa Bowman (1874–1958),

Sometimes in the middle of an animated conversation, and without any apparent cause, he would suddenly commence to stutter so much that it was difficult to understand him [56].

Because his speech impediment tended to disappear in the presence of children, and to return as soon as an adult arrived on the scene, it has been suggested that Dodgson suffered from ‘*a nervous stammer*’, meaning that social anxiety was seen as its underlying cause. A more likely explanation, however, is that he was genetically prone to stuttering, and that nervousness and anxiety got the better of him in the presence of adults, thus aggravating the problem rather than reducing it (which is what happened in the presence of children). As the condition had been present from early childhood onwards, it falls in the category of developmental stuttering (as opposed to acquired stuttering, which usually develops in adulthood). Even now, the exact cause of stuttering is unknown, although it is clear that genetics play an important role. This may well have been the reason why Dodgson’s siblings, Elizabeth , Caroline, and Edwin , also suffered from speech impediments [4], and possibly all of his ten siblings to a greater or lesser degree.³⁰

As Dodgson’s speech impediment also caused him considerable trouble in social life, he resorted to vocal exercises, reading out loud a scene from a play by Shakespeare every day [56]. At some point, he even purchased an *Ammoniaphone* [55], a device designed by the Glaswegian Professor of Chemistry, Carter Moffatt , to improve the quality of his voice (Fig. 4.18).³¹ Disappointed by the results, at the age of 28, Dodgson sought professional help from Dr. James Hunt (1833–1869), at the time the greatest authority alive on stammering , to try out the vocal exercises recommended in his book *Stammering and Stuttering: Their Nature and Treatment* [61]. For a while, he even carried out those exercises together with another sufferer [62]. At the age of 40, he switched to a different system, developed by the speech therapist Dr. J.H. Lewin from Virginia, whose lectures he had attended [63]. Again, he exercised by reading out loud, together with a fellow sufferer. Although he initially showed himself so pleased with the results that he started teaching other sufferers what he had learned, in the

long run this was also of no avail. Therefore, at the age of 41, he went for an interview with Hunt's brother-in-law, Mr. Henry Rivers (1830–1911), who was also a speech therapist, and had taken over Hunt's practice [64]. Again, happy with the initial results, Dodgson exercised together with a fellow sufferer and kept on doing so for several years, meanwhile recommending Rivers to others, including his sisters, for whom he paid the lessons in advance. As he wrote to Rivers on March 29, 1874, he had the intention of '*getting up a system of reading in the family circle*', making a beginning under Rivers' guidance [65]. Whether the plan ever came to fruition is unknown, but Dodgson did make a payment to Rivers for his sisters and kept on practising himself. However, his speech impediment proved resistant to treatment and stayed with him for the remainder of his life.

TRADE MARK

DR. CARTER MOFFAT'S AMMONIAPHONE

Harness Patent

DR. CARTER MOFFAT'S AMMONIAPHONE

is invaluable in all Pulmonary Affections. It is a tube about 25 in. long, constructed of a specially prepared non-spruative metal, with lamellae, closely polished, having patent spring valves. It is charged with a chemical compound, combined so as to resemble in effect that which is produced by the soft balmy air of the Italian peninsula when inhaled into the lungs, hence the term—Artificial Italian Air.

THE VERDICT OF THE DOCTORS.

A. S. KENNEDY, Esq., L.R.C.P., L.R.C.S., &c., writes:—
"14, Conduit-street, London, W., Dec. 23, 1884.
"The two Ammoniaphones that I had from you have given very good results, apart from improved timbre, resonance, and extension of register, which are undeniable. I have found the Ammoniaphone most useful in cutting short catarrhal and laryngeal troubles, and of great benefit in removing huskiness. Several patients have tried the Ammoniaphone at my suggestion and are all pleased with the improvement in their voices."

C. J. BOYD WALLIS, Esq., L.D.S., R.C.S., Eng., 23, Brook-street, Grosvenor-square, London, W., writes, Nov. 21, 1884:—
"I have carefully tested the contents of your Ammoniaphone, and have found it to contain just those ingredients which you have discovered to be present in the air of Italy. The Ammoniaphone forms an excellent inhaler, superior to any other that has come under my notice. I am of opinion that it will be of great value in the treatment of throat and chest affections and in a variable alinate like ours your clever invention will be a desirable remedy to have at hand. Several of my patients have spoken favourably of the Ammoniaphone, and I can fully confirm your Italian air theory."

THE PRESS APPROVES OF IT.
"TRUTH," Nov. 13, 1884:—
"It was, I confess, with profound scepticism that I placed the little silver mouthpiece to my lips, and drew a deep breath. . . . It was not unpleasant, and I persevered, alternating each pull with a good gasp of common air to follow. I had previously been asked to say a sentence or two in my natural voice, and after two or three pulls at the Ammoniaphone, I was requested to repeat the same words without using any extra exertion. I was really startled at the involuntary loudness of my own voice, and a friend who accompanied me, and who was a greater sceptic than myself, fairly burst out laughing at the result. Dr. Carter Moffat smiled benignly, and told me he had now 30,000 persons using the Ammoniaphone with the same results."

DR. CARTER MOFFAT'S AMMONIAPHONE will last for years. Should be used by Actors, Vocalists, Clergymen, Public Speakers, Parliamentary Men, Readers, Reciters, Lecturers, Leaders of Palms, Schoolmasters, Amateurs, Church Choirs, Barbers, and all persons who have to use their voices professionally, or who desire to greatly improve their speaking or singing tones, producing a rich, powerful, melodious voice of extraordinary ringing clearness and range. A poor weak voice becomes rich and massive, while great good is done to the general health.



ST. JAMES'S HALL,
Regent-street.
—
AMMONIAPHONE
CONCERT.
—
M^{me}. MARIE ROZE.
Miss CARLINGFORD.
Mr. W. H. CUMMINGS.
&c. &c.

DR. CARTER MOFFAT'S AMMONIAPHONE

has proved of the utmost value in the treatment of Coughs, Colds, Clerical Throat, Bronchitis, Asthma, Consumption, Aphonia, or Loss of Voice, Deafness resulting from Colds, all Affections of the Throat and Chest, and Sleeplessness. Such ailments may be entirely overcome by means of this simple and beneficent invention.

THE PUBLIC PRAISE IT.
The Very Rev. Dr. TAUGHAN, Dean of Llandaf and Master of the Temple, writes:— Nov. 5, 1884.
"My voice has now nearly recovered its tone, and I have certainly derived benefit, though not suddenly or rapidly, from the use of your Ammoniaphone."
Lady MACFARREN, (wife of Sir G. A. Macfarren, the distinguished President of the Royal Academy of Music) writes:—
"7, Hamilton-terrace, N.W., Oct. 23, 1884.
"I consider the Ammoniaphone to have a wonderfully lasting effect on the vocal organs, and shall have great pleasure in recommending it to such as have weak or relaxed throats; indeed, I have already done so."
Mons. MARIUS, the well-known Comedian, writes:—
"5, Wellington-road, St. John's-wood, N.W., Dec. 27, 1884.
"Dear Sir,—If I have lingered before writing to thank you for the Ammoniaphone, it was because I was determined to give it a fair and exhaustive trial before expressing my opinion. For years I have suffered with my throat, sometimes losing my voice entirely; but since using the Ammoniaphone, although I have had two or three severe colds, I have never lost the use of my voice."
Miss LEONORA BRAHAM, the eminent Operatic Vocalist, writing from the Savoy Theatre, Oct. 31, dates:—
"Having used your Ammoniaphone through a most trying cold, and having thereby obtained great relief, I am anxious to express my admiration of, and thanks for, your truly wonderful invention. This testimony is unsolicited, and is given you to prove the immense value of the Ammoniaphone to all public singers."

"THERE WAS NO DENYING IT."
"PALL MALL GAZETTE," Nov. 7, 1884:—
"Last night St. James's Hall was filled with an intelligent company of musical and scientific people, who were invited to witness the results of the Ammoniaphone. . . . The doctor himself, surrounded by distinguished vocalists, all of whom were Ammoniaphone votaries, explained, instrument in hand, the wonders of his new discovery. . . . Our representative was invited to repeat a few lines in his natural voice, then take some deep inhalations and repeat the same words over again. He did so, and was positively startled at the loudness and sonority of his own voice. The thing seemed absurd; but there was no denying it: the increased volume and improved timbre were too obvious."

DR. CARTER MOFFAT attends daily at the Rooms of the MEDICAL BATTERY CO., 205, Regent-street, London, W., to demonstrate the extraordinary utility of the Ammoniaphone. Write for the "HISTORY OF THE AMMONIAPHONE," an eighty-page treatise, post-free on application.

DR. CARTER MOFFAT AMMONIAPHONE (Harness' Patent) will be sent free by post to any part of the United Kingdom on receipt of P.O.O. or Cheque (crossed "London and County Bank") for 21s., and payable to

C. B. HARNESS, THE MEDICAL BATTERY COMPANY, 205, REGENT-STREET, LONDON, W.

Fig. 4.18 Advertisement for the Ammoniaphone (1885)

4.3.4 Infectious Diseases

Other assaults on Dodgson's health stemmed from the numerous infectious diseases that held 19th-Century England in their grasp. In Western countries in general, those diseases were more prevalent than they are today, and certainly more dangerous, especially to children. Thus, during his high school days, Dodgson spent a year and a half getting through the Lower Middle because of pertussis. As his mother wrote to her sister Lucy,

You will I am sure be as surprised as we are to hear that dearest Charlie really has got the Hooping cough, after having been so proof against the complaint during the whole of his last summer holiday, constantly nursing & playing with the little ones who had it so decidedly. I cannot of course help feeling anxious & fidgetty about him, but at this very favourable time of year for it, I trust the complaint will be of very short continuance & that with care he will get through it as well as our other darlings have done [57].

Mrs. Dodgson had indeed had every reason to be '*anxious and fidgetty*'. Also known as whooping cough, or one-hundred-day cough, pertussis is a highly contagious infectious disease caused by *Bordetella pertussis* (a bacterium rather than a virus) which causes flu-like symptoms followed by fits of coughing that are so severe, that they tend to end in high-pitched gasps for breath (i.e. 'whoops'). As a result of these extreme coughing fits, patients may become exhausted, vomit, and occasionally break their ribs during a fit. As the condition usually lasts for 10 or more weeks, it is no wonder that Dodgson suffered a serious delay with his school work. We do not know whether he went on to have any complications, but it is not uncommon for patients with pertussis to also develop bronchitis, pneumonia, earache, cerebral hypoxia (lack of oxygen to the brain), encephalitis (inflammation of the brain), and/or epileptic seizures. As the causal agent of pertussis was discovered only in 1906,³² in Dodgson's time there was nothing that could be done about it, other than treatment with household remedies, and '*care*', as Mrs. Dodgson had so wisely remarked.

Like all healthy Englishmen, Dodgson also contracted the flu about once a year. His diaries are therefore scattered with brief entries indicating that he had '*a bad cold*' or '*had to stay inside*'. Whether this was always due to influenza is uncertain because an official diagnosis was hardly ever

made and the Influenza virus was only discovered in 1933. Dodgson's diary entries simply indicate that he regularly stayed in, '*not feeling well*', suffering from '*a bad cough*', having '*a sore throat and headache*' or undergoing '*an ague-like feverish attack*'. Other expressions used were '*fever and a sort of ague*', an '*ague-like cold fit*', and a '*feverish cold of ague type*'.³³ One such episode he called '*a sort of rheumatic attack in the neck and shoulders*'. As there is no indication that Dodgson suffered from rheumatoid arthritis or a related auto-immune disease (as those conditions are called today), it would seem safe to assume that this was another flu-like episode. In 1894, at the age of 63, he used the adjective 'rheumatic' again, saying that he had had,

...a dismal time at night with pain in the right shoulder, that I presume is rheumatic, and behind false ribs on that side, suggestive of pleurisy : but it was nearly gone in the morning [69].

Why in this case he thought of pleurisy (i.e. inflammation of the membranes surrounding the lungs) remains unclear. Perhaps he had experienced a type of pain that was new to him, or perhaps there was an unusual combination with dyspnoea. Whatever it was, the day after, the symptoms had apparently taken on a familiar pattern, as he then wrote,

At night my malady took the usual form of 'ague,' and I had the three regular stages [70].

The annually recurring bouts of flu intrigued Dodgson so much, that he wrote a pamphlet about it (Fig. 4.19). It was published in 1881 under the title, *On Catching Cold*, and consisted of extracts from two books by Dr. Thomas Inman (1820–1876), a physician to the Royal Infirmary of Liverpool, plus a note from a book by Dr. James Copland (1791–1870), a Scottish physician and prolific writer on infectious diseases who had spent the early years of his career in the Tropics [71]. A full transcript of *On Catching Cold* can be found in Appendix B. It is not fully clear why Dodgson wrote the pamphlet, and why he took the trouble of publishing it, as it merely reiterated information that was already available from the works by these two authors and lacked any personal perspectives or commentaries. In it, he merely repeated the opinion of the original authors that colds are brought about by sudden changes in temperature, notably

when one passes from a cold environment into a heated room. In that sense, the original authors compared colds to chilblains . In an era predating the discovery of the Influenza virus, the only aspect that could be called ‘revolutionary’, in a sense, was the model’s reversal of the traditional view on catching cold, namely that the hot room that one entered from the cold was seen as the causative factor, rather than the cold itself. The reason why Dodgson wanted to have this argument available in print eludes me. The only reason I can think of, is that he may have wanted to have the pamphlet on hand when discussing the topic with others, so as to back up his argument with ‘written proof’, or at least lend it extra weight by being able to demonstrate the opinion of these two medical experts. What it does show, however, is the extent of Dodgson’s involvement in the topic, as well as his wish to be able to prevent any subsequent bouts of flu .

ON CATCHING COLD.

[Extracted from Dr. Inman's books, 'The Restoration of Health,'
and 'The Preservation of Health.']

'THE most common cause of catarrh is a sudden transition from a moist and cold atmosphere—such as is commonly met with in an “open” English winter—to a hot and dry room; and those people are most subject to “bad colds” who by accident or design have to undergo such transitions. For example, a lady fresh from a ball-room drives home a good long distance on a nasty night in winter. In spite of a comfortable carriage, she respires the cold air of December or January, and arrives at home jaded with dancing and chilled by the night dews. Joyfully she rushes to her comfortable boudoir to find warmth, quiet, and a pleasant nook for chat. But she soon finds that she has “caught a cold”—it may be a fatal one—and then she and her friends lay the blame at the door of the chill on leaving the assembly-room, rather than to the comfort of the chamber of luxury. From long personal experience I would say that no one single

Fig. 4.19 First page of Dodgson's pamphlet, *On Catching Cold* (1881)

In addition to the flu, Dodgson suffered from various other infectious diseases throughout his life. As he wrote at the age of 49, for example, upon staying in for a week after a 23-mile walk on a very hot day, having suffered from what he initially thought to be '*a sort of mixture of sun stroke and ague*',

It has been, I believe, an attack of ‘vesical catarrh ’ with fever as a secondary [72].

And, 3 weeks later,

My feverish symptoms began again on Sunday: so yesterday I sent for Mr. E.L. Hussey, who is now dosing me with quinine . I have no appetite, and Life has become very uninteresting [73].

Judging by the latter passage, Dr. Edward Hussey (1816–1899), a general surgeon, agreed with Dodgson’s own diagnosis of vesical catarrh, which is the old name for cystitis , or bladder infection. Cystitis is caused by bacteria moving up the urethra. Normally these bacteria are flushed out during urination, but, nevertheless, they can stick to the walls of the urethra and bladder and multiply with great speed. As men have longer urethras than women, they tend to be less vulnerable to cystitis, although around 50, they may become somewhat more susceptible. In Dodgson’s case, the cystitis—if diagnosed correctly—may perhaps have been due to dehydration during his long walk in the sun, which probably led to a diminished urine production, and hence a more favourable environment for bacteria to multiply and migrate in the direction of the bladder. Two years on, Dodgson did use the term ‘cystitis’ in his diaries, and wrote that this was indeed what he had meant with ‘vesical catarrh ’:

I have kept my rooms since the middle of Sunday with a sort of ague , with cystitis , like what I had in May 1881. I am trying the same medicines, and today the attack seems to be passing away: but I have had two miserable feverish nights, in a state between waking and sleeping... [74]

Quinine , the medicine he took, may have helped to suppress the symptoms of infection , as it famously does in malaria , but it cannot have provided an actual remedy against the bacteria causing cystitis [75]. Consequently, perhaps, 9 days later, the symptoms had returned. As Dodgson wrote,

I have had a relapse since last Friday, and yesterday was the worst day I have had, even though I had that morning at last called in Mr. Hussey . He approves the quinine, but has chiefly given castor-oil

and black-dose . Tonight I am very thankful to feel really better again [76].

Three days on, Dodgson confirmed that he actually felt so much better that he had started writing again, even though he still had to keep to his rooms [77]. However, a week later, he wrote that he was still '*not well enough*', describing his illness now, in a letter, as '*bilious fever*' [78]. Bilious fever is an old term, derived from Galenic medicine , to designate disorders of the stomach and intestines due to a 'disorder of the bile'. That Dodgson now used this term, rather than 'cystitis ', seems to imply that he had revised his own diagnosis. Moreover, that the castor oil had apparently accomplished what the quinine had failed to do, hints in the direction of a somewhat different problem. Castor oil, also known as *Oleum Palmae Christi* , is a vegetable oil obtained from the seeds of the castor oil plant or *Ricinus communis* [79]. It has many applications, but in medicine, it was primarily used at the time as a laxative. Therefore, Dodgson's real problem may well have been constipation . Since constipation may be complicated by cystitis (especially in females, though) he might have suffered from cystitis after all, albeit in the context of constipation.³⁴

Seven years on, there is another passage in the diaries indicating that, this time around, Dodgson had been keeping to his rooms for 2 weeks, '*suffering from a combination of ague , cystitis and lumbago*' [80], plus a headache [81].³⁵ At the age of 60, he suffered from diarrhoea for 4 or 5 days [82] and at 62 he was on the quinine again, this time because of an '*ague-like cold*' [83].

Other inflammations that he suffered from affected his arms and legs. At the age of 44, he had been '*lame with a gathering on the right heel*' [84], probably due to his many long walks, possibly wearing a new pair of shoes. Two weeks later, despite '*poulticing with spongio-piline in the evening*', he barely succeeded in '*bearing a boot long enough to attend University Sermon*' [85, 86]. When he was 56, there was a problem with the right knee. It was diagnosed as 'synovitis ' by the navy surgeon, Dr. Robert Walter Doyne (1857–1916), who painted the knee with iodine and bandaged it from the foot upward [87]. Dodgson stayed in bed and on the sofa for more than 3 weeks and eventually got an elastic knee-cap. Nonetheless, it took 5 weeks before he could walk more or less comfortably again.

Synovitis is an inflammation, usually sterile in nature, of the membranes that line a joint, in this case the knee joint. It typically presents with pain, redness, warmth, and swelling, the latter due to synovial fluid collection. Whether Dodgson actually suffered from this is impossible to tell, as there are various other knee-joint effusions that present with pain and swelling (such as osteoarthritis, as pointed out by Goodacre [24]). The diaries provide no descriptions of inflammation (i.e. redness, local warmth, and fever) even though Dodgson himself, in a letter to Lucy Walters (b. 1856), speaks of the knee being inflamed:

For the last 3 weeks I have spent most of my time on my sofa, with a bandaged knee, which chose to be inflamed [88].

Even if it had been synovitis —or osteoarthritis for that matter—there had been no logical reason to paint the knee with iodine, as in both cases a wound or infected area on the outside is lacking. Even so, the ‘synovitis’ was a huge problem to Dodgson, who had to postpone his habitual walks and had to stay close to home. He was incapacitated for 6 weeks; after that the knee still troubled him so much, that after 3 months he visited the world-famous surgeon and pathologist, Sir James Paget (1814–1899), for advice. Unfortunately, there is no surviving indication of Paget’s diagnosis.

A year later, Dodgson exclaimed,

Just now I am the victim of various maladies! A boil on the left wrist, which has lasted about a month, is scarcely healed yet. A suppurating ‘pile’ drove me to Mr. Sherwood on Saturday, but is now better. The ‘synovitis,’ which a year ago was in the right knee, has now attacked the left, and today I have begun a course of bandaging and iodine. Also today I saw ‘fortifications ’: but no headache followed [89].

This time, the knee problem kept him inside for a week or more. Dodgson applied a process to ‘*get the extra fluid absorbed*’ but does not tell us what that involved. As regards the boil on the left wrist, this was probably an inflamed area of skin due to the bacterium *Staphylococcus aureus* , which may enter the skin through a tiny wound or even a hair follicle. The suppurating ‘pile’ must have been due to the pus characteristic of boils; this

is usually treated with a small incision to remove the pus. It is probably that what Dodgson referred to in his diary, when he subsequently wrote,

Mr. Sherwood came to perform a small surgical operation, by removing the small fleshy excrescence for which I had consulted him. He applied cocaine first, to numb the nerves. The pain was severe, but only lasted a few seconds [90].

As is usual during such procedures, Dr. Arthur Sherwood (1852–1923) probably applied cocaine as a local anaesthetic, thus avoiding any effects on the brain and the rest of the body. Antibiotics may also be necessary under such circumstances, but those were not available in Dodgson's days and, apparently, he recovered very well without them.

A year on, he was again '*laid up for a week with synovitis in left knee (probably rheumatic)*' [91]. Why this time he considered the knee problem to be 'rheumatic' in nature remains unclear. Another year on, he recorded yet another episode of 'synovitis' [92]. It was in the left knee again, which he now treated with '*much bandaging and painting with iodine*' [93]. As this was of no avail, he discarded the bandaging, kept the leg in a straight position and treated it with rest and a daily coating of iodine-paint [94]. Being of no avail either, he and Dr. Brooks then tried massaging it [95]. As this did not suit the knee either, he went back to the iodine and moderate walking [96]. In all, it took him 3 months to heal, during which time he lived the life of '*a hermit*' [97]. The 'fortifications' mentioned before were probably migraine equivalents—but we will come back to that later.

4.3.5 Medicine Use

Throughout his life, Dodgson used various medicines to treat or prevent medical problems, some of which now strike us as relatively straightforward, and others as rather obscure. For example, in 1863 and 1871, Dodgson had himself vaccinated against smallpox. That was a wise thing to do, since smallpox (a highly contagious disease caused by the *variola* virus) was a devastating disorder that caused three out of ten victims to die, and often left those who survived with such widespread skin lesions and scars, that they were marked for life (Fig. 4.20).³⁶ I say 'was', because during the 1960s and 1970s the world was rid of smallpox after a global eradication programme orchestrated by the World Health

Organisation (WHO) . In Dodgson's time, however, it was still a very grave threat, with very grave consequences, designated fittingly as '*the most dreadful scourge of the human species*' [99].³⁷

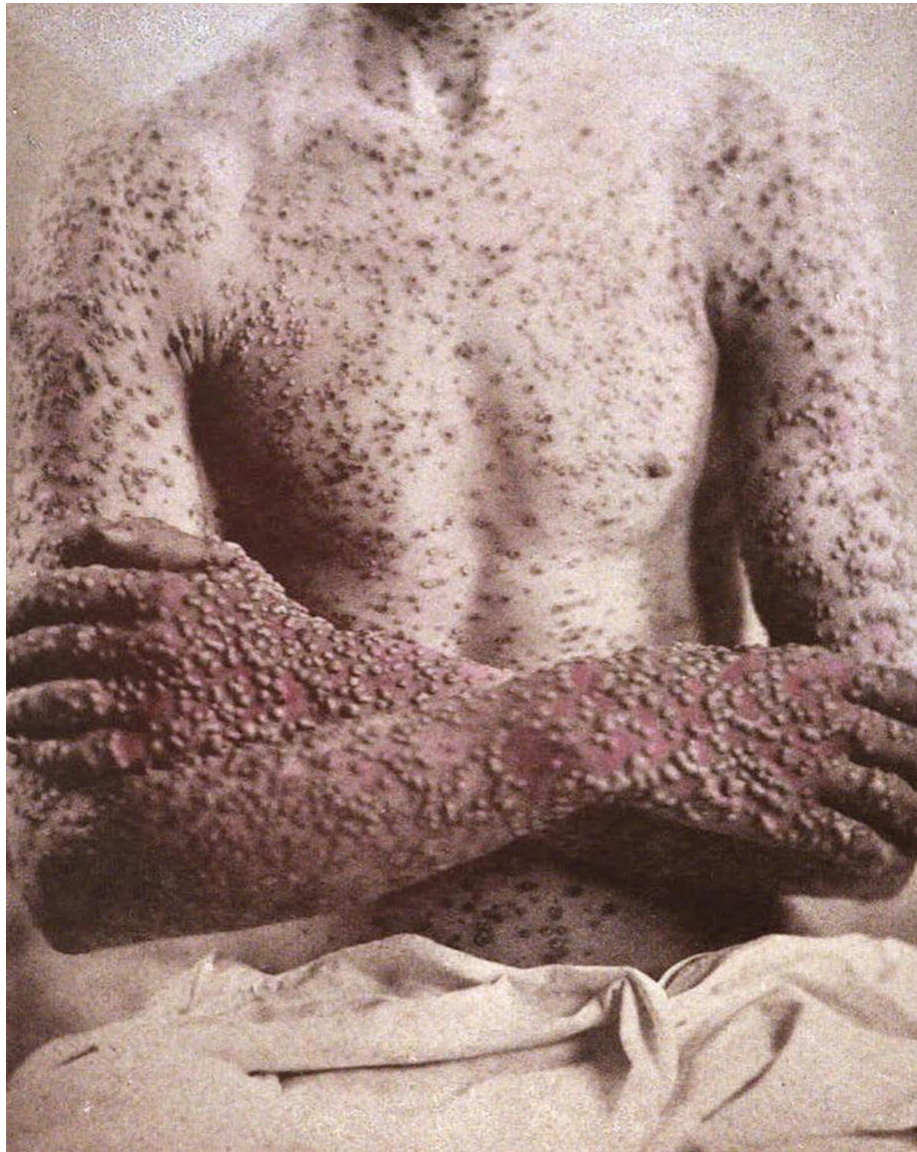


Fig. 4.20 A case of smallpox, from *Photographic Illustrations of Skin Diseases* by George Henry Fox (1886)

At the age of 35, Dodgson procured a salve from Parisian nuns to treat *tic-doloreux*, as he called it [100]. Tic doloureux is an old collective term for facial pain, or *neuralgia faciei* , of which even during the first half of the 19th Century no less than nine variants had been described [101]. Undoubtedly the most striking type, still known today, is trigeminal

neuralgia , a pain syndrome characterised by attacks of stabbing pain in the part of the face that is innervated by the trigeminal nerve, usually on one side. It is known as one of the most excruciatingly painful conditions one can suffer from. It may be accompanied by facial spasms, and those affected may even be driven to acute suicidal behaviour . On the basis of Dodgson's diaries it is unlikely that this was what he meant with the term *tic-doloreux*. Trigeminal neuralgia is in fact so characteristic and so overwhelming in its presentation, that neither he nor people in his surroundings would have been able to miss it, had he really been suffering from it.

In his travel diary of 1867, there is an oft-recounted story about the perseverance with which he sought to obtain the salve from the nuns, who were famous for their home-made medicinal product, but refused to sell it because it was meant to be given away for charity. On the basis of that story it has been assumed that Dodgson himself suffered from neuralgia at the time, although he does not say so in his diary entries. It may therefore have been out of medical curiosity that he sought to obtain the salve, or to save it for future needs, or else with the intention of passing it on to someone else suffering from *tic-doloreux*. In all, it seems more likely that he suffered from a different variant of *neuralgia faciei* from time to time, since a year before his visit to the Parisian nuns, he used the term 'neuralgia' to designate a combination of neck and facial pain, which he had apparently grown used to, and which in that particular instance appears to have been connected with tooth decay:

My old enemy, neuralgia, has shifted its quarters from the neck to the face, where it gave me several days of considerable pain, partly I fancy owing to the weather, and partly to a hollow tooth. However summer weather has come, the tooth stopped, and the neuralgia gone for the present, I am happy to say. I interested myself in making out from my Cyclopedia its exact name, which I believe to be 'neuralgia suborbitalis ' [102].

If Dodgson's self-diagnosis was correct, this means that he had suffered from pain spreading through the cheek, upper lip, nose, and lower eye lid (although slightly different patterns are also possible) [103]. The cause of this pain would have been stimulation of the upper maxillary nerve, most

likely due to an infection . The temporal relation with the dental caries seems to suggest that the latter was the actual cause.

That Dodgson sometimes sought to help other people suffering from facial pains is something we know from a letter to Elizabeth Hussey (1810–1896), whom he told that,

My sister recommends highly for neuralgia , Shirley’s Magic Crystal (to be rubbed over the affected part). Any chemist would get it [38].

Although *Shirley’s Magic Crystal* was apparently a popular medicine during the 1880s, I was unable to retrieve any references to this intriguing substance. What this passage appears to imply, though, is that Dodgson himself had found no need to use it, or else he might as well have shared his own experiences with it with Mrs. Hussey .

When he was 61, Dodgson reported that he had stayed in for a week, taking a ‘*medicine to set the liver etc. straight*’, prescribed to him by Dr. Walter Brooks (1859–1942), a physician practising at Oxford [104]. Which medicine it was, is unknown, and whether there actually was a liver problem is impossible to establish with hindsight.

As we saw, at least some medicines taken by Dodgson were homeopathic in nature. At the age of 50, he had detected an oval patch of pink, shining skin under one arm. The homeopath, Dr. Edward Shuldhham (1837–1924), diagnosed it as *tinea* (i.e. *tinea versicolor*), a discoloration of the skin which we now know to be caused by a fungus from the genus *Malassezia* (which was not yet known at the time). Dodgson tells us that Shuldhham advised sulphurous acid to treat it. What was probably meant by it, was an *aqueous solution* of sulphurous acid ,³⁸ which is often referred to with the same name. Historically, sulphurous-acid solutions have often been used as disinfectants, so we can therefore safely assume that Shuldhham advised it to prevent the patch of skin from becoming infected. However, Dodgson doubted the diagnosis made by Shuldhham and, instead, thought he was suffering from ‘*some form of erythema* ’ [105]. Therefore, rather than following up his advice, he treated himself with *Graphites* (a homeopathic solution based on carbon, to be used internally) and *Calendula* (common marigolds, a medicinal herb, to be used externally).

In 1888 he consulted another homeopath, most likely Dr. James Compton Burnett (1840–1901), who thought ‘*the spleen out of order, and prescribed for that, as the probable cause of eczema and varicosis*’ [106]. It is a rather cryptic sentence, but a little further on in his diary, Dodgson elucidates that Burnett gave him a prescription for ‘*Rubia Tinctoria (Madder), 5 drops, of strength ϕ , in water, night and morning*’. Whether he actually suffered from eczema or varicosis is unclear. Equally unclear is whether he tried the medicine that Burnett prescribed. He sounds somewhat taken aback when he observes that, ‘*It is a drug introduced by him: at least it is not named in the books*’ [106].³⁹

4.3.6 Self-Doctoring

Having a considerable interest in medical topics, Dodgson possessed a modest library of medical books, as well as a set of bones, which he apparently used to teach himself some basic principles about the human body. Not only was he into mainstream medical thinking but, as we saw, also into homeopathy, not hesitating to put into practice things that he had learned. After his death, in his suite in Oxford two boxes of bottles were found, containing homeopathic and herbal remedies [55]. According to Wakeling, he often carried one of those boxes around for use in emergency situations [108]. Thus, he was once able to treat his child friend Agnes Hull (1867–1936) with *Calendula* after she had cut her foot on a broken bottle at the beach, 2 weeks later supplying her with *Nux vomica* (a homeopathic solution based on poison-nut) to treat her ‘sick headache’, and meanwhile providing *Calendula* lotion to one of her relatives, who suffered from blistered feet [109, 110]. In fact, he was so much into alternative medicine that he sought the acquaintance of various homeopaths, collected reference books on homeopathic and herbal remedies and gave some of his books away to family members and friends, sometimes along with a box of remedies.

4.3.7 Injuries

Throughout his life, Dodgson sustained various injuries. Once, while visiting his child friend Ethel Arnold (1866–1930), he was bitten by the family’s dachshund [4]. That put him at risk for tetanus and rabies, both prevalent and potentially life-threatening infectious diseases, but nowhere do we read that he attracted either condition. At the age of 33, he made an

attempt to skate and then fell, cutting open his forehead ‘*by hurrying too precipitately*’. A year later, after climbing Great Gable in the Lake District in icy cold weather, he came down with a swollen face. The condition was called ‘neuralgia’ by his nephew, Stuart Dodgson Collingwood [16]. Neuralgia is a rather general medical term, used to designate a stabbing, burning pain due to irritation of the nerve endings. As Dodgson’s diary indicates that he indeed suffered from facial pains for at least 4 days in the aftermath of the journey, it is certainly possible that his nephew’s diagnosis was correct [111, 112]. Two months before that, Dodgson had recorded to be suffering from neuralgia, too, and therefore had had to miss the Freemasons’ Fête. As he did not elaborate on his symptoms, it is unsure whether this was a similar condition.

At the age of 54, he suffered from lower back pain, to which he referred as ‘*lumbago, or sciatica, or perhaps a mixture of the two*’. Whether he consulted a physician is unknown, but because of it, he did not dare to go out into the cold [32]. In fact the lumbago was something that plagued him for much of his life, forcing him to work and write in his characteristic upright position.

4.3.8 Loss of Consciousness

At the age of 59, Dodgson once hit his head and lost consciousness for an hour. This event has had huge consequences for the ideas, built over time, regarding the perceptual aberrations described in the *Alice* book. As Dodgson wrote in his diary,

I must have fainted just at the end of morning chapel, as I found myself, an hour afterwards, lying on the floor of the stalls; and had probably struck my nose against the hassock, as it had been bleeding considerably. It is the first time I fainted quite away. I sent for Dr. Brooks. I had some headache afterwards, but felt very little the worse. It is of course possible it may have been epilepsy and not fainting: but Dr. Brooks thinks the latter [113].

Six days on, the idea of an epileptic seizure had nevertheless taken root, judging by the next entry on this topic:

Nearly well again, though still not free from headache. Dr. Brooks now thinks it was an epileptic attack, passing off into sleep [114].

Whether it was really Dr. Brooks who now favoured an epileptic origin, or rather Dodgson himself, is hard to tell. Why, after all, would Dr. Brooks have changed his opinion when there had been no other after-effects than the headache? With that question in mind, it is interesting to compare the two passages above with the one below, from a letter written by Dodgson on April 26, 1891, in which he does not even seem to debate the diagnosis of epilepsy anymore:

More than 2 months ago, I woke up one morning from an uneasy dream , saying to myself ‘how very uncomfortable the pillow is!’ and found myself lying on the floor, up in the stalls of the Cathedral. I wouldn’t believe it at first, but thought I was still dreaming : but in a few moments I was broad awake, and found it really was so. I was lying in a pool of blood, having bled profusely from the nose, which no doubt had received a heavy blow in my fall (in fact the doctor said the bones were loosened, and would take several weeks to get set firm again), and had been lying there exactly an hour. I remembered distinctly the reader of morning-prayers having come to within a few words of the end: and I find I remained kneeling when the others left the building. The 2 tutors, who went last, noticed that I did not get up, but concluded I was only going on in private prayer a little longer than usual, and thought no more of it: and the vergier never noticed I had not gone out, but barred the doors, and left by another door. So I had the place all to myself, to sleep off the attack (epileptic, no doubt), and then unbarred the doors and let myself out. Luckily I met no one on my way back to my rooms, for I was a pretty figure! With my face and my shirt-front all covered with blood. My doctor found me to be out of health generally - at least the digestion was out of order - and this may have caused the attack. Anyhow, the result has been a great deal of headache, and unfitness for brain-work... the doctor thought I had been doing too much brain-work, and sitting up too late... I have been ‘taking it easy,’ now, for a good while, and my headaches are getting fewer, and my brain recovering its usual power... [115]

In all, it took Dodgson more than 10 months to get rid of the headaches. In his letter to Edith Blakemore (1872–1947), cited above, he had shown himself quite confident that he was on the mend, but it was only on Christmas Day of the same year that he was able to report that, ‘*My headaches have vanished, and my full powers of work have returned*’ [94].

Whether the cause of his troubles had indeed been epilepsy, as he had now come to believe, has been much debated. For one thing, it is uncommon to suffer from headaches for so long in the aftermath of an epileptic seizure. In all, 81% of all headaches following an epileptic seizure last less than 6 hours, and only 8% last longer than 24 hours [116]. Therefore, if the pain had had anything to do with the incident in the Cathedral, it would have been much more likely that it had been due to the traumatic injury (possibly sustained by other factors, such as physical or psychological stresses) than to epileptic activity. The reason why Dodgson himself kept on clinging to a diagnosis of epilepsy may have been that his brother, Skeffington Dodgson (1836–1919), as well as his nephew, Stuart Dodgson Collingwood, had suffered from it, and that he had witnessed the latter getting a fit on at least one occasion [117]. In addition, he had witnessed seizures in several other people. The first time, an acquaintance had been seized with a fit near the Anatomy School. Like any other lay person who has ever witnessed such a savage event, Dodgson could not help but feel ignorant and useless under the circumstances, and therefore resolved to ‘*make a point of reading some book on the subject of emergencies*’ [118]. Being a man of his word, he went on to order *Hints for Emergencies*, the first in a long line of medical books that would find their way into his bookcase. When years later, he witnessed another man suffering a seizure in the tramway-car at Ryde, he was able to be of some sort of assistance. In accordance with the old adage, ‘*See one, do one, teach one*’, he subsequently recorded in his diary that he ‘*was able to be of some little use, being now fairly experienced in that kind of fit*’ [119]. In a similar vein, 2 years on, he recorded how he

...came in for one of my usual ‘accidents’ - a woman in a fit (slight epilepsy): I made them lay her flat etc. and helped to lift her off the beach into a bathing-machine, by which time she was beginning to come round [120].

A year later, he had again been able to be of help:

While I was [at the Queen's Hotel], a Mr. Dixon, staying at the Hotel, fell down outside in a fit of epilepsy : I was glad to be able to be of some use in attending him, and consoling his terrified wife [121].

This time, only 2 weeks went by before he ran into yet another case:

After calling on the Baynes, I came (with my usual luck) on a case of 'fits' - a little boy, in a semi-unconscious state: he showed that he felt it every time the cold water, with which they were bathing his head, touched him. I was a little puzzled as to what it was: but as, in another minute, he was quietly walking home, I suppose it was merely a case of 'petit mal ' [122].

Whether all these cases were genuine instances of epilepsy is hard to tell with hindsight, but they indicate that, over the years, Dodgson had indeed witnessed up close various seizures which were at least suggestive of epilepsy. It is no wonder, therefore, that this possibility sprang to his mind in connection with his own 'attack'. However, an even more important reason for him to think of this possibility may have been a prior incident which he had experienced himself at the age of 53, described by him as follows:

On the morning of Dec. 31st or late New Year (I think it was) I had an attack ('epileptiform' - Dr. Morshead called out) which left me with a sort of headache , and not feeling my usual self, for a week or 10 days. Edwin heard me, and he and Fanny came in: and they got Dr. Morshead and Dr. Stedman (!). It seems to have been but a mild attack, and I don't think it's in the family. Stuart Collingwood's case is I think, in family, as Charles Collingwood has had similar attacks [123].

From this brief description it is hard to tell—again—whether this had been a genuine epileptic seizure . It is well possible, however, since this time around, several witnesses had been present, plus two physicians who had arrived at the scene immediately afterwards. Still, whatever it was that these

people had witnessed, remains unclear. To some biographers, the time of the attack, somewhere around New Year's Eve, has raised suspicions of an alcohol withdrawal seizure . Could that have been the reason why Dr. Ernest Morshead (1851–1912) had called it '*epileptiform*' rather than '*epileptic*'? Or could Dodgson have misunderstood him, or not fully grasped the difference between 'epileptic' and 'epileptiform'? The difference is important, since the term 'epileptiform' is usually reserved for cases that resemble epilepsy but raise doubt as to their actual nature (e.g. in hysteria or dissociation). Moreover, during the 19th Century, it was also used sometimes when an attack was unilateral, with involvement of only one arm and one leg, rather than both arms and both legs [124]. In the absence of any further evidence, the incident may also have been caused by a vasovagal collapse or by orthostatic hypotension , to mention two additional possibilities. In the absence of any positive findings on an EEG , a diagnosis in accordance with current biomedical practice would have been hard to make anyway, especially since vasovagal collapses may sometimes present with symptoms that are indistinguishable from those of an epileptic seizure (hence the use of the name *convulsive syncope* in such cases [125]).

In conclusion, there are some indications that Dodgson may have experienced two tonic-clonic seizures throughout his life, although neither account is in any way convincing, and definite proof is lacking. Nonetheless, whatever it was, he himself was convinced that it had been epilepsy, adding at a later date that both 'attacks' had taken place in the winter, and ascribing the first one at least partly to '*a cold day in London*' [126].

4.3.9 Migraine

The reason why the possibility of epilepsy in Dodgson's medical history is considered important, is that this neurological disorder can be preceded or accompanied by perceptual phenomena characteristic of Alice in Wonderland syndrome. Another candidate for such phenomena is migraine, a condition Dodgson also thought to be suffering from. The reason for this was that, throughout his life, he had suffered several times from fortifications , or flickering scotomata , as they are also called, in the visual field. The first time that he mentioned something awry with his eye, was at the age of 33. In an enigmatic entry, he wrote,

Consulted Mr. Bowman, the Oculist, about my right eye: he does not seem to think anything can be done to remedy it, but recommends me not to read long at a time, nor on the railway, and to keep to large type by candlelight [127].

Dr. William Bowman (1816–1892) was a seasoned ophthalmic surgeon, even at the relatively young age of 40, when he examined Dodgson. Had he been confronted with a common eye disorder such as floaters , nearsightedness, farsightedness, conjunctivitis, uveitis, cataract , glaucoma , corneal abrasion, a dislocated lens, diplopia or even one of the neurological disorders that were already known as possible causes for visual trouble by then, he would certainly have recognised it, and in some of these instances have been able to do something about it, too, or at least have given some specific advice. Instead, the advice he gave was so unceremoniously commonplace, that it would seem that he hardly believed the problem to be in the eye.

The advice to refrain from reading too long, and from reading a small letter type in the absence of sufficient light, sounds more like the type of advice physicians would have given at the time to someone on the brink of ‘asthenia ’, ‘nervous exhaustion ’ or whatever term was used for the typical Victorian ailments affecting the weak and feeble. Likewise, the advice not to read on the railway would seem to stem from the concern that this rough, new travel might pose serious health risks, especially to vulnerable people, and to those who were already sick or weak. We are speaking here of the year 1856, when steam trains, like all technological novelties, raised concerns about consumer safety. At the time, the authoritative medical journal *The Lancet* even called for independent studies to chart the hazards of this new acquirement. The results confirmed what many Victorians had already feared:

Many of the submissions viewed the motion of the train, the shaking and the vibrations, as a source of harm. And some parts of the body, such as the uterus and the brain, were seen as more sensitive than others. Pregnant women and sick people were thought to be at greatest risk. Submissions made to *The Lancet* can be read as an attempt to make sense of these anxieties [128].

As one contributor to *The Lancet* wrote at the time,

The stout, easy-going, lethargic traveller, I notice, bears continuous locomotion far better than the spare, nervous, irritable man [129].

Thus, there is reason to believe that Bowman's advice to Dodgson was aimed at alleviating a mental rather than an ophthalmologic problem. The nature of that problem was kept under wraps by Dodgson, but as he frequently exhausted himself by reading and writing at night, and in his diaries referred time and again to his sleepless nights, these circumstances may perhaps offer a logical explanation for Bowman's course of action.

However, as noted by Podoll and Robinson, it may also have been the case that Dodgson suffered from a negative spot in his visual field, due to a migraine without headache [130]. Whether this is true is unknown, but if it was, it would probably have been something that Bowman was not familiar with. Although historical accounts of the visual phenomena accompanying migraine date back to Hippocrates (c. 460 BC–370 BC), it was not until 1870 that Hubert Airy (1838–1903), a British physician who himself suffered from migraine, published a paper that would eventually lead to a wider recognition of this phenomenon [131]. There is certainly more to be said about Podoll and Robinson's analysis of the situation (see Chap. 7), but I believe that they were right in assuming that Dodgson may have suffered from migraine, as in later years he did report a recurring phenomenon that positively hints in that direction: starting in 1885, 20 years *after* the publication of *Alice's Adventures in Wonderland*:

In the morning I experienced, for the second time, that odd optical affection of seeing moving fortifications, followed by a headache [132].

With the term '*moving fortifications*', Dodgson can hardly have meant anything other than a geometric visual hallucination, also known as a flickering scotoma, or fortification of Vauban,⁴⁰ which is characteristic of migraine. It presents as an extremely bright zigzag line, most often white, though sometimes multicoloured, which begins near the fovea in one half of the visual field, and then spreads towards the outer limits of the same hemi-field. It may have a pulsating, flickering quality, and while moving towards the outer limits may temporarily leave a blind spot in its wake, a kind of

‘hole’, so to speak, in the patient’s visual field. As Dodgson had mentioned that he had experienced these fortifications ‘*for the second time*’, he may have meant that he had also experienced them at the time he visited Dr. Bowman, although it would seem somewhat odd to refer to something as being experienced a second time when the first time had been so long ago. Here, again, it is unfortunate that Dodgson’s diary entries were so brief, and that we cannot be sure how thorough he was in documenting his own medical history. His final remark, that the fortifications were ‘*followed by a headache*’, fits the hypothesis that this was an instance of migraine, even though headaches come in many varieties, and the entry does not provide us with any details about the particular kind that Dodgson had.

However, there is more. Three years on, we find another passage describing this archetypal phenomenon:

This morning, on getting up, I experienced that curious optical effect, of ‘seeing fortifications,’ discussed in Dr. Latham’s book on ‘bilious headache.’ In this instance, it affected the right eye only, at the outer corner; and there was no headache [133].

The book Dodgson referred to in this entry, was *On Nervous or Sick-Headache* by Dr. Peter Latham (1832–1923), a small volume presenting two lectures that the physician had given at Addenbrooke’s Hospital [134]. In it, Latham described ‘*nervous headache, sick-headache or bilious headache*’, three terms that he used interchangeably to designate ‘*attacks commencing with mistiness before the eyes, and succeeded by unilateral frontal pain, nausea and vomiting*’ [134]. Quoting Airy, the physician who had experienced migraine himself and with whom Latham had corresponded on the subject, he reproduced the latter’s drawings of fortifications in his book (Figs. 4.21 and 4.22) and went on to describe these visual phenomena in detail, including the loss of vision in one hemi-field by which they may be followed. Regarding the origin of the attacks, Latham noted that the majority of persons he had examined had been anaemic, with a ‘*want of tone about the pulse*’, and of ‘*a nervous temperament*’. As he continued,

Their brains are excitable, their senses acute, and their imaginations free. The attacks are induced by prolonged mental work, protracted

mental excitement, or any intense strain on the feelings, such as grief, anxiety , passion, etc. Bodily fatigue, late hours, loss of sleep, the depression which follows over-excitement, a debauch, etc., are all predisposing causes [134].

Latham concluded that a '*general exhaustion of the powers*' could produce a '*lowering of the tone of the body*', a contraction of the vessels of the brain and, due to interference by the sympathetic nervous system, a subsequent widening of the vessels *and* the headache . He hypothesised that '*gastric derangement*' might also be a cause, hence his use of the term '*bilious headache* ', which implied a derangement of the abdominal organs. As a kind of afterthought, he even noted that he saw a relationship, '*though happily a distant one*', with epilepsy —a relationship many clinicians today still consider of relevance, even though Latham's other ideas have long since been discarded.

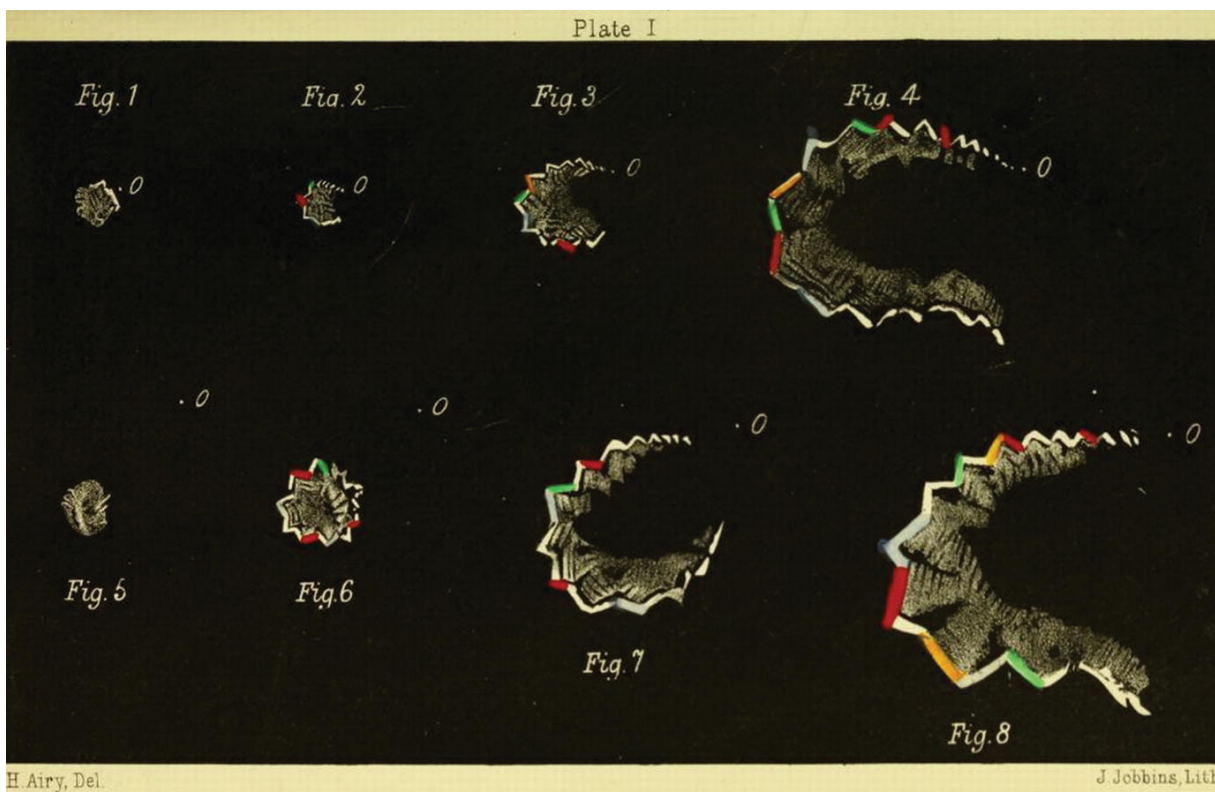


Fig. 4.21 Fortifications characteristic of the migraine aura; Plate I from Hubert Airy's paper (1870)

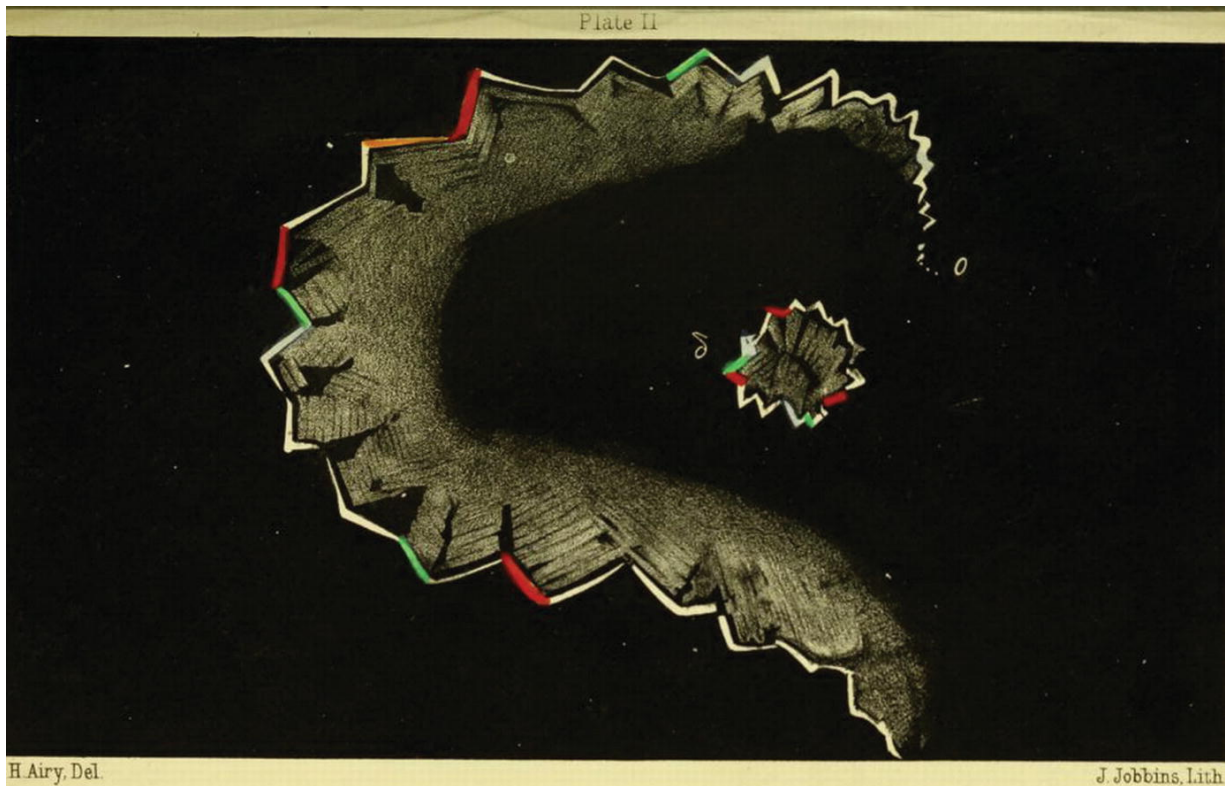


Fig. 4.22 Fortifications characteristic of the migraine aura; Plate II from Hubert Airy's paper (1870)

Considering the clinical descriptions of fortifications and their relation with 'bilious headache' in Latham's book, it is no wonder that Dodgson recognised himself in what he read, including, perhaps, the alleged predilection of the disorder for people with '*excitable brains*' and '*free imaginations*' who work themselves up to a state of '*bodily fatigue*' and '*loss of sleep*' due to '*late hours*'. Today migraine and its accompanying aura phenomena are primarily attributed to a neuropathological process called *spreading depression*, and the condition is conceptualised as thoroughly neurological in nature, with psychological factors acting as mere risk factors for initiating or aggravating an attack. Although, in Dodgson's time, explanatory models were of course different, descriptions of the disorder's accompanying visual phenomena were very accurate.

As Dodgson indicated in his diaries, after the attack of June 1888, three more instances followed. Thus, half a year later, he reported,

Again experienced the optical 'fortifications .' It began with a distinct loss of a large piece of the area of vision of the left eye, the

‘blind’ patch being in the right-hand corner, just where, directly afterwards, the ‘fortifications’ appeared [135].

Although he does not mention it, apparently there had been no headache this time. This was also true the following year, when he wrote,

Also today I saw ‘fortifications’: but no headache followed [89].

And then again, 2 years later:

Last evening I had another experience of ‘seeing fortifications’ [136].

In all, Dodgson thus reported 5 instances of migraine equivalents in 7 years, meanwhile suggesting that they had been preceded by another one which (due to lack of information in any of the primary sources) may or may not have been the reason why he had consulted Dr. Bowman decades before. It is not entirely clear how many of these 6 instances had been followed by a headache, but the fortifications themselves indicate with sufficient accuracy that they had been genuine instances of migraine. As a consequence in 1952, the American neurologist Caro Lippman (1886–1954) saw reason to suggest that Dodgson had suffered from not only migraine but also accompanying aura phenomena, and that these experiences might well have acted as a source of inspiration for the peculiar perceptual distortions described in *Alice’s Adventures in Wonderland* [137]. Although possible, Dodgson’s diaries fail to confirm that he had ever experienced aura phenomena. That is to say, except—perhaps—6 days prior to his last experience of ‘*seeing fortifications*’, when he had asked himself whether he had perhaps witnessed some sort of telepathy. I already quoted the relevant passage before (in the section on Dodgson’s membership of the SPR) but do so again here for easy reference:

At the evening service of Christ Church, a curious thing happened, suggestive of ‘telepathy.’ Before giving out the second hymn, the curate read out some notices. Meanwhile I took my hymn-book, and said to myself (I have no idea why) ‘it will be Hymn 416,’ and I turned to it. It was not one I recognised as having ever heard: and, on looking at it, I saw ‘it is very prosaic: it is a very unlikely one.’

And it was really startling, the next minute, to hear the curate announce ‘Hymn 416!’ [36]

Obviously, it may all have been some sort of strange coincidence, this business with Hymn 416, and Dodgson somehow knowing that that would be announced. Alternatively, it may have been an instance of *déjà vu*, meaning that Dodgson had merely heard the curate announce this particular Hymn, and simultaneously had the overwhelming sensation of having heard this before (or having known this already, in which case we would have to speak of *déjà connu*). People unfamiliar with *déjà* experiences often look back at such instances and try to come to grips with what had happened, by constructing some logical explanation. As it is impossible to tell whether this was what Dodgson had done, perhaps we should take his word for the order of events as described by him. Nevertheless, considering the fact that he had had this experience relatively shortly before his final instance of seeing fortifications, and considering the almost stereotypical way in which he explained it, I think we should keep an open mind for the possibility that this might have been a *déjà* experience of some sort.⁴¹

With this observation, our overview of Dodgson’s medical history comes to a close. We will return to this in Chap. 7 but now, as promised, I would like to focus on several aspects of Dodgson’s life that have remained unclear. When dealing with the biographical material, several loose ends had been left dangling. Since they may bug you as much as they have me whilst working on this book, I would like to make them explicit and present them here for what I think they are: questions without answers. Nevertheless, I think these questions are certainly worth addressing.

4.3.10 Questions Without Answers

I feel totally out of my depth here in my capacity as a 21st-Century continental psychiatrist, but perhaps I am not the only one who does not fully grasp how certain things fit together concerning Dodgson. Foremost, while researching his life and work, I came to ask myself what on earth we can make of his involvement with the Liddell household. What had been going on there and what had attracted these people so much to each other? I can see what had attracted them at the outset, but the question remains of how and why they ended up in such a knotty situation. And, intimately

connected with that question: Why was this family-oriented, successful, marriageable man never openly in a relationship?

Upon accepting his academic position at Christ Church, Dodgson was obliged to take a vow of celibacy. This constitutes a fairly plausible reason why, for several years at least, he had not gone on to find a partner. However, that is not where the story ends. After 4 years, he would have had to enter priesthood but, for some reason or another, he postponed that. When, finally, 5 years had passed and he was about to lose his position because of all the stalling, he was summoned by Dean Liddell to make up his mind. Telling the Dean—anxiously, no doubt—that he was not ready for it, he received word that the matter would be brought before the Electors, and that the certain outcome would be that he would have to quit his job. It is not hard to imagine how badly Dodgson must have slept that night. However, one day later, to his and everyone's surprise, that ominous perspective had mysteriously evaporated. Against all rules and regulations, the Dean allowed him to remain in his position as tutor of mathematics for the remainder of his working life, thereby making him the only academic in the history of Christ Church to have ever gained permission to do so.

What could have been the reason for that? What could have made Dodgson so special in the eyes of the Dean that the notoriously strict rules of Christ Church did not apply to him? Had Lorina Liddell—the Dean's wife—been behind this? Considering Dodgson's tangled relation with her husband, the latter could hardly have been expected to dream this up by himself. On the contrary, this would have been an outstanding opportunity for him to get rid of the man who, at work at least, continued to give him so much trouble.

Consequently, from that point on, Dodgson would have been free to get married. Moreover, he could have quit his job at the university at any time, especially after the revenues from his books had started to outgross his monthly pay check. Nevertheless, he did not marry—and he did not quit. He remained a bachelor and stayed in the same working position throughout his life without ever seeking a promotion, remaining attached with numerous strings to the university life that he was so ambiguous about and—tellingly perhaps—staying in close proximity of the Deanery. When, many years later, the Dean retired, Lorina Liddell went on the hunt for another house and, allegedly, consulted Dodgson about houses in the vicinity of Guildford, where he had previously bought one for his sisters to

live in and where he quite regularly stayed himself. What was it between the two of them that seemed to make them keep on circling each other, despite their public fall-out?

In the light of this, I have been asking myself the following question. Could it perhaps have been that, during those early years, Dodgson had envied the Dean's position at home, and silently longed to take his place as the husband of one of the most gorgeous looking and most sparkling women of all Oxford who, on top of that, was the mother of some of the most adorable children he had ever laid eyes on? And could it be that, in his secret, scheming fantasies—if he ever had them, of course—he himself had sometimes been confused about whom to court in this ready-made family full of attractive women? Others have often zoomed in on his love for little girls, making Alice—or perhaps her slightly elder sister—the alleged object of his desire, whereas his contemporaries gossiped behind his back that he secretly fancied the governess. While, theoretically, Dodgson may indeed have been plotting to wait until either of Lorina's daughters would turn 14 and then ask for her hand in marriage (not an unusual procedure at the time), what about his feelings for Lorina herself? This is merely a question, no more than that. However, in the light of Collingwood's suggestion that he had once been rejected by a woman he had loved, the recurring theme in his poems of 'love betrayed', together with the whole bizarre situation at the Deanery (where he already fulfilled the role of a surrogate parent), why would the Dean's wife not have been the one to which he had felt attracted? On this topic, however, I must admit that my lack of familiarity with the British way of life does not help me at all. What I do get (even though the finer subtleties elude me) is that the rules of engagement in Victorian England made it nigh impossible for a person of lower social status, such as Dodgson (who was a mere tutor and, moreover, of humble descent), to strike up an acquaintance with someone with higher social status, such as the Dean—let alone with his wife, who must have been totally off-limits for someone like him if it hadn't been for the special relationship he had forged with her children, which, in itself, was already curious enough.

Now, if you thought that Alice was the only one whose adventures got '*curiouser and curiouser*' as she went along, please bear with me, since Dodgson's were hardly conventional either. We already noted how remarkable it was that he had been allowed to walk in and out of the Dean's

house on a daily basis for over a year, occupying the man's place in the family as a quasi-parental figure. However, what is even more remarkable is that the Dean eventually tolerated him for 50 years in his vicinity, despite the flak he had to take from him at work, which often amounted to sheer character assassination [14]. Strikingly, when the end came for Dodgson, he and the Dean died only 4 days apart and, most strangely, they were commemorated in a single sermon at Christ Church Cathedral by Dean Francis Paget (1851–1911), who had taken over Liddell's position at the university [14]. So here is another question that I cannot simply brush aside: What was it between these two men, who must have been barely able to stand each other, that their lives remained so absurdly intertwined—from the moment they had met, until the moment they both died? Is this whole story just a sign to remind us that life has a sense of irony? Or was there something more to the interdependent relation of these two men—something that goes totally over our heads?

And then there was, of course, Alice (Figs. 4.23 and 4.24): she is the one who has been targeted more than anyone as Dodgson's alleged object of desire—at least since the Victorian notion of the inherent innocence of the love for children had been trampled by psychoanalytic thought. What was that all about? There is no denying that Dodgson had loved Alice. However, as we saw, there is no concrete evidence that he fancied her in a romantic or sexual way. Long after the happy days at the Deanery were over, Dodgson had written to Lorina Liddell that it had felt, at the time, as if the situation would last forever [31]. Clearly, he had had fond memories of Alice and of the episode in itself—so much so, that he had wished it had never come to an end. And yet, as early as 1865 when the *Alice* book came out, to the Liddell family it had already been '*a postscript to the friendship rather than a celebration of it*' [34]. As we saw, soon thereafter the boat trips with the children had been annulled and, eventually, contacts with the family had petered out. Although throughout his life Dodgson did his best to stay in touch with Lorina and her daughters, tellingly, Dean Liddell's posthumously written biography—commissioned and overseen by Lorina—does not mention him a single time. In yet another attempt to wipe out all memories of Dodgson, Lorina urged Alice to destroy all the letters she had ever received from him [34]. By that time Alice had already kept her distance from Dodgson for many years. Moreover, throughout her life, she had remained reluctant to tell her story. When finally she gave in for once,

in 1932, it was under the pressure of public demand, at the centenary of Dodgson's birthday (Fig. 4.25). What she told the press back then was a carefully rehearsed story, so as to meet the expectations of the public. But how did she really look back on her time with Dodgson? And how must it have been for her to be associated all her life with the character in this book that everybody knew and loved? Since actions speak louder than words, we should probably not go over the news clippings of that time, in which she was celebrated as '*the real Alice*', exactly the way the audience wished to see her—especially since, a few years beforehand, she had sold the original *Alice* manuscript for a record price of £15,400 to help out her bankrupt son [34]. Even though blood is supposedly thicker than water, that does not tell us what it must have meant to Alice to abandon this unique document, and whether she was glad to be rid of it, or now missed this token of one '*golden afternoon*' gone by.



Fig. 4.23 *Alice Pleasance Liddell as beggar-child, aged six, photographed by Charles Dodgson (1858)*



Fig. 4.24 *Alice Pleasance Liddell*, aged 18, photographed by Charles Dodgson (1870)



Fig. 4.25 *Alice Pleasance Liddell Hargreaves, aged 80, photographed by W. Coulbourn Brown (1932)*

All in all, I have no real clue as to what it had been between Alice Liddell, her parents, and Charles Dodgson. If there is one thing that I have learned during my years in psychiatry, it is that reverence and hate are two sides of the same coin. Whether people adore each other or feel repelled, it is the passion they put into their actions that counts. Since it is passion that makes things worthwhile anyway, the time that these people spent together

must have been of premium quality. The way they loved and fought, laughed and cried, made each other blossom and sometimes suffocated each other—it was all very passionate. Nevertheless, that does not explain why they behaved the way they did; but, then again, who am I—as a 21st-Century continental psychiatrist—to understand such a thoroughly Victorian and British affair? Luckily, my task is to explore this fascinating perceptual disorder called Alice in Wonderland syndrome—to which we shall now return—and not to disentangle the convoluted relationships of Charles Dodgson and the Liddells.

As we saw, despite these difficult relationships, Dodgson had succeeded in living a meaningful, highly successful life up until the relatively old age of 65 years—although not without having to battle a sizeable number of afflictions, some of which were minor ailments whereas others were relatively severe by any standards. Among all those afflictions, there were at least two that qualify as potential causes for the symptoms that we now recognise as being part of Alice in Wonderland syndrome, namely, migraine and epilepsy. However, in either case it remains unsure whether the disorder affected him *before* he wrote *Alice's Adventures in Wonderland*, and it certainly remains doubtful whether he ever actually suffered from epilepsy. To establish whether there might have been different reasons for him to experience symptoms of Alice in Wonderland syndrome, Chaps. 5 and 6 will focus on the syndrome itself, providing an overview of its wide variety of symptoms, its underlying mechanisms (in so far as they are known) and any associated medical conditions. After that, as promised, Dodgson's health issues will be revisited in Chap. 7, where I seek to shed more light on the issue of whether his various health problems justify the assumption that he had grafted Alice's mind-blowing perceptual experiences on those of his own.

References

1. Wilson R (2008) Lewis Carroll in Numberland. His fantastical mathematical logical life. Penguin Books, London
2. Reed L (1932) The life of Lewis Carroll. W. & G. Foyle, London
3. Dodgson CL (1999) Diary entry of November 9, 1865. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson, volume 5. September 1864 to January 1868. The Lewis Carroll Society, Luton

4. Lennon FB (1945) *The life of Lewis Carroll. Victoria through the looking glass*. Simon & Schuster, New York
5. Dodgson CL (1993) Diary entry of August 27, 1855. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 1. January to September 1855*. The Lewis Carroll Society, Luton
6. Dodgson CL (1993) Diary entry of September 4, 1855. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 1. January to September 1855*. The Lewis Carroll Society, Luton
7. Dodgson CL (1994) Diary entry of March 6, 1856. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856*. The Lewis Carroll Society, Luton
8. Dodgson CL (1994) Diary entry of March 8, 1856. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856*. The Lewis Carroll Society, Luton
9. Dodgson Family Collection, Leach K (2009) *In the shadow of the dreamchild. The myth and reality of Lewis Carroll*, 2nd edn. Peter Owen, London
10. Dodgson CL (1997) Diary entry of May 12, 1863. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 4. May 1862 to September 1864 and a reconstruction of the four missing years*. The Lewis Carroll Society, Luton
11. Dodgson CL (2004) Diary entry of December 9, 1891. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892*. The Lewis Carroll Society, Luton
12. Plazzi G (2013) Dante's description of narcolepsy. *Sleep Med* 14:1221–1223 [[PubMed](#)]
13. Dante A (1472) *La comedia di Dante Alleghieri*. Johann Numeister and Evangelista Angelini da Trevi, Foligno
14. Day D (2015) *Alice's adventures in Wonderland decoded*. Doubleday Canada, Toronto
15. Dodgson CL (1979) Letter to Tom Taylor, June 10, 1864. In: Cohen MN (ed) *The letters of Lewis Carroll*. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 1, ca. 1837–1885. Oxford University Press, New York
16. Dodgson Collingwood S (1899) *The life and letters of Lewis Carroll*. T. Fisher Unwin, London
17. Waldron HA (1983) Did the Mad Hatter have mercury poisoning? *Br Med J* 287:161
18. Gardner M (2015) *The annotated Alice. 150th anniversary deluxe edition*. Expanded and updated by Burstein, M. W.W. Norton, New York
19. Carroll L (1890) *The nursery Alice*. Macmillan, London
20. Torrey EF, Miller J (2014) *Violence and mental illness: what Lewis Carroll had to say*.

Schizophr Res 160:33–34
[PubMed]

21. Holley AJF, Greenwood PJ (1984) The myth of the mad March hare. *Nature* 309:549–550
[PubMed]
22. Rossetti WM (1906) Some reminiscences of William Michael Rossetti. Charles Scribner's Sons, New York
23. Wakeling E (ed) (2007) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 10. Complete index. Reconstruction of the first two missing volumes. The Lewis Carroll Society, Luton
24. Goodacre SH (1972) The illnesses of Lewis Carroll. *Practitioner* 209:230–239
[PubMed]
25. Fine EJ (2013) The Alice in Wonderland syndrome. In: Finger S, Boller F, Stiles A (eds) Literature, neurology, and neuroscience. Neurological and psychiatric disorders. Elsevier, Amsterdam, pp 143–156
26. Davies MJ (2010) Alice in Waterland. Lewis Carroll and the river Thames in Oxford. Signal Books, Oxford
27. Nichols C (2014) Alice's Wonderland. A visual journey through Lewis Carroll's mad, mad world. Race Point Publishing, New York
28. Lovett Stoffel S (1997) Lewis Carroll and Alice. Thames & Hudson, London
29. Dodgson CL (1993) Diary entry of August 4, 1855. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 1. January to September 1855. The Lewis Carroll Society, Luton
30. Dodgson CL (1999) Diary entry of May 11, 1865. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 5. September 1864 to January 1868. The Lewis Carroll Society, Luton
31. Leach K (2009) In the shadow of the dreamchild. The myth and reality of Lewis Carroll, 2nd edn. Peter Owen, London
32. Wakeling E (ed) (2004) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
33. Dodgson CL (2001) Diary entry of January 16, 1873. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 6. April 1868 to December 1876. The Lewis Carroll Society, Luton
34. Woolf J (2010) The mystery of Lewis Carroll. Discovering the whimsical, thoughtful, and sometimes lonely man who created "Alice in Wonderland". St. Martin's Press, New York
35. Dodgson CL (2003) Diary entry of August 13, 1881. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton

36. Dodgson CL (2004) Diary entry of September 6, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
37. Sidgwick H, Johnson A, Myers FWH, Podmore F, Sidgwick E (1894) Report on the Census of Hallucinations. In: Proceedings of the Society for Psychical Research. Volume 26. Part 10. Kegan Paul, Trench, Trübner, London, pp 25–422
38. Wakeling E (ed) (2003) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
39. Dodgson CL (2004) Diary entry of November 17, 1889. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
40. Dodgson CL (2004) Diary entry of December 13, 1889. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
41. Dodgson CL (2005) Diary entry of May 9, 1895. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
42. Dodgson CL (1999) Diary entry of July 15, 1867. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 5. September 1864 to January 1868. The Lewis Carroll Society, Luton
43. Wakeling E (1999) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 5. September 1864 to January 1868. The Lewis Carroll Society, Luton
44. Dodgson CL (1995) Diary entry of July 31, 1857. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 3. January 1857 to April 1858. The Lewis Carroll Society, Luton
45. von Krafft-Ebing R (1886) *Psychopathia sexualis. Mit besonderer Berücksichtigung der conträren Sexualempfindung*. Verlag von Ferdinand Enke, Stuttgart
46. Greaves LM, Barlow FK, Lee CHJ, Matika CM, Wang W, Lindsay CJ, Case CJB, Senqupta NK, Huang Y, Cowie LJ, Stronge S, Storey M, de Souza L, Manuela S, Hammond MD, Milojev P, Townrow CS, Muriwai E, Satherley N, Fraser G, West-Newman T, Houkamau C, Bulbulia J, Osborne D, Wilson MS, Sibley CG (2017) The diversity and prevalence of sexual orientation self-labels in a New Zealand national sample. *Arch Sex Behav* 46:1325–1336 [[PubMed](#)]
47. Green RL (ed) (1953) The diaries of Lewis Carroll. Volume I. Cassell & Company, London
48. Green RL (ed) (1953) The diaries of Lewis Carroll. Volume II. Cassell & Company, London
49. Dodgson CL (1997) Diary entry of August 14, 1864. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 4. May 1862 to September 1864 and a reconstruction of the four missing years. The Lewis Carroll Society, Luton
- 50.

- Dodgson CL (1997) Diary entry of June 28, 1864. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 4. May 1862 to September 1864 and a reconstruction of the four missing years. The Lewis Carroll Society, Luton
51. Dodgson CL (1997) Diary entry of March 6, 1864. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 4. May 1862 to September 1864 and a reconstruction of the four missing years. The Lewis Carroll Society, Luton
 52. Dodgson CL (1999) Diary entry of May 21, 1867. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 5. September 1864 to January 1868. The Lewis Carroll Society, Luton
 53. Chapman DL (1994) Sandow the magnificent. Eugen Sandow and the beginnings of bodybuilding. University of Illinois Press, Chicago
 54. Dodgson CL (2003) Diary entries of May 31 through June 5, 1882. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
 55. Cohen MN (1995) Lewis Carroll. A biography. Macmillan, London
 56. Bowman I (1899) The story of Lewis Carroll. Told for young people by the real Alice in Wonderland. E.P. Dutton, New York
 57. Wakeling E (ed) (1993) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 1. January to September 1855. The Lewis Carroll Society, Luton
 58. Dodgson CL (1994) Diary entry of June 18, 1856. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856. The Lewis Carroll Society, Luton
 59. Carroll L (1871) Through the looking-glass (and what Alice found there). Macmillan, London
 60. Dodgson CL (1979) Letter to Arthur Gilkes, April 25, 1891. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 2, ca. 1886–1898. Oxford University Press, New York
 61. Hunt J (1854) Stammering and stuttering. Their nature and treatment. Longmans, Green, London
 62. Dodgson CL (1997) Diary entry of October 29, 1862. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 4. May 1862 to September 1864 and a reconstruction of the four missing years. The Lewis Carroll Society, Luton
 63. Dodgson CL (2001) Diary entry of June 5, 1872. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 6. April 1868 to December 1876. The Lewis Carroll Society, Luton
 64. Dodgson CL (2001) Diary entry of September 18, 1873. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 6. April 1868 to December 1876. The Lewis Carroll Society, Luton
 - 65.

Dodgson CL (1979) Letter to Henry Rivers, March 29, 1874. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 1, ca. 1837–1885. Oxford University Press, New York

66. Nicholls A (2000) Fenland ague in the nineteenth century. *Med Hist* 44:513–530
[\[PubMed\]](#)[\[PubMedCentral\]](#)
67. Wakeling E (ed) (2005) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
68. Dodgson CL (1979) Letter to Mrs. B. Dyer, March 7, 1887. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 2, ca. 1886–1898. Oxford University Press, New York
69. Dodgson CL (2005) Diary entry of September 14, 1894. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
70. Dodgson CL (2005) Diary entry of September 15, 1894. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
71. Dodgson CL (1881) On catching cold. University Press, Oxford
72. Dodgson CL (2003) Diary entry of May 27, 1881. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
73. Dodgson CL (2003) Diary entry of June 14, 1881. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
74. Dodgson CL (2004) Diary entry of July 10, 1883. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
75. Shanks GD (2016) Historical review: problematic malaria prophylaxis with quinine. *Am J Trop Med Hyg* 95:269–272
[\[PubMed\]](#)[\[PubMedCentral\]](#)
76. Dodgson CL (2004) Diary entry of July 20, 1883. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
77. Dodgson CL (2004) Diary entry of July 23, 1883. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
78. Dodgson CL (2004) Diary entry of July 30, 1883. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
- 79.

Tunaru S, Althoff TF, Nüsing RM, Diener M, Offermanns S (2012) Castor oil induces laxation and uterus contraction via ricinoleic acid activating prostaglandin EP₃ receptors. *Proc Nat Acad Sci U S A* 109:9179–9184

80. Dodgson CL (2004) Diary entry of October 31, 1890. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
81. Dodgson CL (1979) Letter to Skeffington Dodgson, November 8, 1890. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 2, ca. 1886–1898. Oxford University Press, New York
82. Dodgson CL (2005) Diary entry of September 23, 1892. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
83. Dodgson CL (2005) Diary entry of March 24, 1894. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
84. Dodgson CL (2001) Diary entry of May 5, 1876. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 6. April 1868 to December 1876. The Lewis Carroll Society, Luton
85. Dodgson CL (2001) Diary entry of May 6, 1876. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 6. April 1868 to December 1876. The Lewis Carroll Society, Luton
86. Dodgson CL (2001) Diary entry of May 21, 1876. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 6. April 1868 to December 1876. The Lewis Carroll Society, Luton
87. Dodgson CL (2004) Diary entry of March 29, 1888. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
88. Dodgson CL (1979) Letter to Lucy Walters, March 17, 1888. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 2, ca. 1886–1898. Oxford University Press, New York
89. Dodgson CL (2004) Diary entry of September 2, 1889. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
90. Dodgson CL (2004) Diary entry of September 9, 1889. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
91. Dodgson CL (2004) Diary entry of March 25, 1890. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton

92. Dodgson CL (2004) Diary entry of November 25, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
93. Dodgson CL (2004) Diary entry of December 6, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
94. Dodgson CL (2004) Diary entry of December 25, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
95. Dodgson CL (2004) Diary entry of February 9, 1892. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
96. Dodgson CL (2004) Diary entry of February 15, 1892. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
97. Dodgson CL (2004) Diary entry of January 18, 1892. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
98. Cohen MN (1979) The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 1, ca. 1837–1885. Oxford University Press, New York
99. Jenner E (1801) On the origin of the vaccine inoculation. D.N. Shury, London
100. Dodgson CL (1999) Diary entry of September 13, 1867. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 5. September 1864 to January 1868. The Lewis Carroll Society, Luton
101. Society for the Diffusion of Useful Knowledge (1839) The penny cyclopædia of the Society for the Diffusion of Useful Knowledge, vol 15. Charles Knight, London
102. Dodgson CL (1979) Letter to Lucy Lutwidge, April 2, 1866. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 1, ca. 1837–1885. Oxford University Press, New York
103. Rowland R (1838) A treatise on neuralgia. S. Highley, London
104. Dodgson CL (2005) Diary entry of May 10, 1893. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
105. Dodgson CL (2003) Diary entry of November 8, 1882. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
106. Dodgson CL (2004) Diary entry of October 27, 1888. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892.

The Lewis Carroll Society, Luton

107. Clarke JH (1902) A dictionary of practical materia medica, vol 2. The Homœopathic Publishing Company, London
108. Wakeling E (2015) Lewis Carroll. The man and his circle. I.B. Tauris, London
109. Dodgson CL (2003) Diary entry of September 1, 1879. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
110. Dodgson CL (2003) Diary entry of September 17, 1879. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
111. Dodgson CL (1994) Diary entry of August 23, 1856. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856. The Lewis Carroll Society, Luton
112. Dodgson CL (1994) Diary entry of August 26, 1856. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856. The Lewis Carroll Society, Luton
113. Dodgson CL (2004) Diary entry of February 6, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
114. Dodgson CL (2004) Diary entry of February 12, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
115. Dodgson CL (1979) Letter to Edith Blakemore, April 26, 1891. In: Cohen MN The letters of Lewis Carroll. Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green. Volume 2, ca. 1886–1898. Oxford University Press, New York
116. Hughes JR (2005) Did all those famous people really have epilepsy? *Epilepsy Behav* 6:115–139
[\[PubMed\]](#)
117. Dodgson CL (2003) Diary entry of June 23, 1881. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
118. Dodgson CL (1994) Diary entry of March 1, 1856. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856. The Lewis Carroll Society, Luton
119. Dodgson CL (1995) Diary entry of August 28, 1875. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 3. January 1857 to April 1858. The Lewis Carroll Society, Luton
120. Dodgson CL (2003) Diary entry of August 8, 1877. In: Wakeling E (ed) Lewis Carroll's diaries.

The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton

121. Dodgson CL (2003) Diary entry of July 21, 1882. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. The Lewis Carroll Society, Luton
122. Dodgson CL (2003) Diary entry of August 3, 1882. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 7. January 1877 to June 1883. Luton, The Lewis Carroll Society
123. Dodgson CL (2004) Diary entry of January 20, 1886. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
124. Arts N (1996) Introduction. In: Arts N (ed) John Hughlings Jackson. Selected writings, vol 1. Nijmegen, Arts & Boeve, pp vii–xiv
125. Loesch AM, Becker A, Noachtar S (2013) Syncope: there is more than haemodynamic failure. *BMJ Case Rep* bcr2013010347
126. Dodgson CL (2005) Diary entry of December 25, 1892. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 9. July 1892 to December 1897. The Lewis Carroll Society, Luton
127. Dodgson CL (1994) Diary entry of January 17, 1856. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 2. January to December 1856. The Lewis Carroll Society, Luton
128. Hickie C (2009) A fatal form of contentment. *Perm J* 13:88–91
[[PubMed](#)][[PubMedCentral](#)]
129. Acton W (1862) The influence of railway travelling on public health: personal experiences of an habitual traveller. *Lancet* 79:210–211
130. Podoll K, Robinson D (2008) *Migraine art. The migraine experience from within*. North Atlantic Books, Berkeley
131. Airy H (1870) On a distinct form of transient hemiopia. *Philos Trans R Soc Lond* 160:247–264
132. Dodgson CL (2004) Diary entry of May 23, 1885. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
133. Dodgson CL (2004) Diary entry of June 12, 1888. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
134. Latham, P.W. (1873). *On nervous or sick-headache. Its varieties and treatment*. Deighton, Bell, Cambridge
- 135.

- Dodgson CL (2004) Diary entry of December 3, 1888. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
136. Dodgson CL (2004) Diary entry of September 12, 1891. In: Wakeling E (ed) Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson. Volume 8. July 1883 to June 1892. The Lewis Carroll Society, Luton
137. Lippman CW (1952) Certain hallucinations peculiar to migraine. *J Nerv Ment Dis* 116:346–351
[\[PubMed\]](#)
138. Podoll K, Robinson D (2002) Splitting of the body image as somesthetic aura symptom in migraine. *Cephalalgia* 22:62–65
[\[PubMed\]](#)
-

Footnotes

¹ Robinson Duckworth, however, later recalled, “*I rowed stroke and he rowed bow in the famous Long Vacation voyage to Godstow, when the three Miss Liddell’s were our passengers, and the story was actually composed and spoken over my shoulder for the benefit of Alice Liddell, who was acting as ‘cox’ of our gig. I remember turning around and saying, ‘Dodgson, is this an extempore romance of yours?’ And he replied, ‘Yes, I’m inventing as we go along.’*” [\[1\]](#)

² Tellingly, perhaps, in his diaries Dodgson initially stubbornly misspelled his name as ‘Tenniell’.

³ Tenniel even went on to illustrate Dodgson’s next book, *Through the Looking-Glass*, but after that, he threw the towel into the ring, citing Dodgson’s ‘pretentiousness’ and ‘obstinacy’ as obstacles to further collaboration. Upon hearing that Harry Furniss (1854–1925) had agreed to illustrate the subsequent project, *Sylvie and Bruno*, he warned the latter by saying, ‘*I’ll give you a week, old chap; you will never be able to put up with the fellow any longer. He is impossible!*’ Dodgson, meanwhile, told Furniss that, ‘*...with the exception of Humpty Dumpty he did not like Tenniel’s drawings*’ [\[2\]](#).

⁴ This second ‘first edition’ was printed in 1865, although the title page bore an imprint of 1866.

⁵ Although this was by no means the actual first copy of *Alice’s Adventures in Wonderland* handed out by Dodgson, it was obviously a unique gift to give and to receive.

⁶ In Victorian England, girls generally needed to be chaperoned after they had turned 14 years old.

7 As remarked by David Day , the *Alice* story follows an even older basic pattern, derived from one of the oldest recorded myths in the entire world literature [14]. It is the myth of the Mesopotamian goddess Inanna , who descends into the underworld, where people go to after they have died. The motif is better known from the classical story of Persephone , which begins with her and her older sister Demeter in an idyllic meadow, follows Persephone after her fall into a deep fissure that leads into the underworld, and sees her return safely into the arms of Demeter in the end.

8 Incidentally, in Dodgson's time, the expression 'Mad as a March hare' was as common as 'Mad as a hatter', perhaps being a corruption of Erasmus ' expression *mad as a marsh hare* [18, 21].

9 As proposed by Day [14], an alternative reading is that the Mad Hatter stood for the Cambridge Christian Socialist, Charles Kingsley (1819–1875), the March Hare for the Christian Socialist theological writer, Julius Charles Hare (1795–1855), and the Dormouse for another famous Christian Socialist, John Frederick Denison Maurice (1805–1872), together symbolising a 'mad symposium of quarrelling Cambridge philosophers belonging to the Christian Socialist Party'. Similarly, from the vantage points of different modes of conceptualisation, he mentions alternative possibilities for the gnostic tradition, mythology and philosophy.

10 To whom the Duchess (Fig. 4.12) refers is unknown, but Tenniel drew her in the likeness of *The Ugly Duchess* , a satirical portrait of the Duchess of Corinthia and Tyrol by the Flemish artist Quinten Matsijs (1466–1530) (Fig. 4.13) [2].

11 This also applied to his academic output, which consisted mainly of compilations and elaborations of the work of the Greek mathematician Euclid (c. 325–c. 270 BC), alternated with displays of sheer brilliance in high-ranking scientific journals, which appeared to stem from flashes of creative insight rather than from a particular line of research he had been working on. Some examples of this are a method for computing the day of the week for any given date, and an extension of the Venn diagram from three fields to more than ten [1].

12 According to Collingwood [16], Dodgson kept a journal from the age of 10; the last entry was written a few days before his death in 1898.

13 Even though Dodgson abhorred self-aggrandisement, refused interviews for magazines, and habitually turned down people seeking his autograph, many entries in his diaries are about meeting important others, and about obtaining their permission to photograph them or get their autograph. He was very successful at this. Had he lived in our time, he would certainly have been a well-connected player on Social Media.

14 The reason why Dodgson had a habit of wearing gloves is not fully understood. The etiquette of glove-wearing was intricate in the Victorian era, and the circumstances under which one put them on and off were stipulated by numerous social codes. Dodgson may well have worn them as a fashion accessory and because of the social mores that they stood for. He started wearing them at a young age, as indicated by a letter home, written by him at the age of 17 years, in which he reported that he had bought a new pair. Lovett Stoffel [28] suggests that an additional reason may have been, at a later age at least, that his hands were often stained black by the chemicals he used while practising his 'black art', photography. As far as we know, he did not wear gloves to hide any skin diseases.

15 This double standard is remarkable, since Dodgson himself greatly enjoyed impressing others with his authorship. Apparently, the crucial aspect here was whether he was in control or not when others were let in on his 'secret'.

16 According to Leach [31], this leaves open the possibility that Dodgson had romantic and/or sexual affairs with adult women, even though there is no evidence for this in any of the primary sources available.

17 Hydrophobia was the old name for rabies ; in 1883, Dodgson wrote a letter to the *St. James's Gazette* detailing his opinion on how to deal with this life-threatening disorder in dogs as well as in humans [32].

18 A tiny island in the South Atlantic Ocean, with living conditions so harsh, that Dodgson advised the British government to move the entire population to South Africa.

19 In fact, Dodgson considered his own work of such grave importance that he sent Harry Furniss , his illustrator for *Sylvie and Bruno* , a lengthy nondisclosure agreement for him to sign, comparable to the agreements sent nowadays by Hollywood studios to actors in big-budget films. Furniss refused to sign the document and was astonished to find that Dodgson reacted by sending him the manuscript all cut up in horizontal strips of four or five lines each, randomly reassembled and pasted on sheets of paper, and marked with mysterious numbers, letters and hieroglyphs to indicate their proper order. Meanwhile, he forbade Furniss to let anyone see him work on the manuscript, including his wife, whom he told that she should consider herself '*the most privileged woman in the world, for she knew the man who knew his (Lewis Carroll's) ideas*', adding that, '*it was sufficient for her to gaze at [her husband] outside of [his] studio with admiration and respect, as the only man, besides Lewis Carroll himself, with a knowledge of the latter's works*' [4].

20 The first major project organised by the SPR was the *Census of Hallucinations*, a field study designed to find out whether visions of dying people were just that, or whether there was something more to them. People had been telling each other stories of the kind in which their uncle in Ceylon had appeared to them in a vision, extending an arm, and whispering inaudibly as if for the last time, and the letter they had received a month later, containing news of his sad demise in the jungle on the same day, or about the crew mate who had died, and after his burial at sea had been seen treading the waves, or about the dog whose barking had been heard every day, even though it had been dead for many years. To investigate the veracity of such claims, Sidgwick and his team mobilised 410 volunteers and sent out questionnaires all over the UK, eventually collecting no less than 17,000 individual responses, the affirmative ones of which were followed up by face-to-face interviews with one of the volunteers. After dreams and other dubious cases had been excluded, Sidgwick's committee calculated that 9.9% of the population in the UK had experienced one or more hallucinatory episodes during their lives. As the Committee's primary focus was on signs of life from beyond, they then selected all reports of individuals who had died within 12 hours before or after they had appeared in any of the respondents' hallucinations. After excluding all accounts that reeked of foreknowledge, the Committee was eventually left with 350 first-hand reports of death-related visions. They ran their statistical tests and established that this number was 440 times higher than could have been expected on the basis of chance alone. Consequently, the Committee concluded that, '*between deaths and apparitions of the dying person a connexion exists which is not due to chance alone*' [37]. As this conclusion was supported by similar results by sister groups from France, Germany, and the USA, there appeared to be little room for doubt that there was more between Heaven and Earth than traditional science was inclined to admit.

21 Dodgson wrote—or merely intended to write—a paper under the title, *Miracles —Why have they ceased?*—however, whatever came of it is unknown [38].

22 Among the early members of the SPR were the co-founder of Darwin's evolutionary theory, Alfred Russel Wallace (1823–1913), the physicists Oliver Lodge (1851–1940) and Heinrich Hertz (1857–1894), the physicist and Nobel laureate Pierre Curie (1859–1906), and his wife Marie Curie-Skłodowska (1867–1934), the first woman to win a Nobel prize, and the only person to ever win twice; the French physiologist and Nobel laureate Charles Richet (1850–1935), who so firmly believed in the existence of ectoplasm, that he directed all his efforts at unravelling the nature of this mysterious substance; the psychologists Gardner Murphy (1895–1979) and William James (1842–1910), Britain's prime minister Arthur James Balfour (1848–1930) and his brother Gerald William Balfour (1853–1945), the poet William Butler Yeats (1865–1939), and the author Samuel Langhorne Clemens, better known as Mark Twain (1835–1910). The Society's group of corresponding members, moreover, included Sigmund Freud (1856–1939), Carl Gustav Jung (1875–1961), Cesare Lombroso (1836–1909) and Pierre Janet (1851–1947), all leading European psychiatrists at the time.

23 Another aspect is food. Being a modest eater, perhaps food was something that he did not care too much about, but most people keeping diaries will probably reflect now and then on an exceptionally good meal, a fancy new restaurant being opened, or the efforts of their host to prepare something special. Not Dodgson. He would sometimes recount with whom he had shared a meal and mention occasionally what had been on the table (such as meat or mutton chops, or soup), but he never provided any further details.

24 There were exceptions, of course. Especially during the final decade of his life, Dodgson allowed himself to let off steam in his diaries once in a while, calling someone whom he had just met, ‘*rather “out of his mind”*’, calling a dentist he had consulted ‘*a humbug*’, and fuming about his publisher that he had just received, ‘*A further proof of incompetent managing at Macmillan’s—I had written on the 8th, to beg they would send me 12 copies of Sylvie and Bruno to Oxford as soon as possible. Copies were ready on Thursday morning. This is Friday evening, and none have come. Mr. Craik is the only senior acting partner: and he is an amateur: Macmillan’s sons are young. I fear the Firm is going down, and I may have to find another publisher*’ [39–41].

25 For the purpose of a screenplay or a Hollywood movie rather than the context of the present book, one might perhaps be tempted to connect the dots in this brief, yet striking passage; however, there is hardly any need for this, as too much has already been written on Dodgson’s alleged sexual preferences in the virtual absence of any primary sources documenting them. One only needs to Google ‘Carroll’ in combination with ‘paedophilia’ or ‘pornography’ to stumble upon thousands of posts commenting on these issues. The vast majority of them lack any historical accuracy and merely repeat what others have said before, or lose themselves in insinuating daytime reveries. Yet even among scholars, this theme has yielded a remarkable rift, forcing them to side either with those who turn a blind eye to any hint or suggestion of sexuality in Dodgson’s life, or with those who would gladly fill in whatever information is lacking to arrive at sometimes truly colourful conclusions.

26 Leach [31] suggests that something of a collective cover-up may have taken place and goes on to demonstrate, simply by pointing out the birth dates of Dodgson’s many ‘child friends’, that he did hang out with sexually mature girls and women, visiting them alone, having them at his home unchaperoned, taking them out to the theatre or to an artist’s studio, and occasionally letting them sleep in his studio at Oxford, at the family home at Guildford, or in summer houses that he rented. Especially after his death, and in 1932, at his birthday centenary, women reported themselves in droves to share their stories with the media, suggesting that they had been one of the few who had been friends with Dodgson’s as a child, even if they had met him only once on a train, or merely thought that they had seen him. So enticing was the prospect of being associated with this Victorian VIP that they sometimes went to great lengths to polish their stories and did not shrug from inventing new ones. As Leach demonstrates convincingly, women who thus reminisced about their ties with their illustrious friend were not afraid to lie about their age, stating that they had been 10 or 11 years when they had been with him, whereas their birth dates show that they had actually been in their late teens or in their twenties. She goes on to argue that all information pointing in the direction of any romantic or sexual interest on Dodgson’s side was suppressed by his fellow Victorians, giving later biographers a hard time adjusting the public image thus created. The question of whether this collective censoring, if it did take place, had been a matter of white lies, meant to protect the reputations of all involved while nothing untoward had happened, or whether they had the purpose of hiding something that was not allowed to see the light of day, is something that we may never be able to answer. The reason why none of Dodgson’s former child friends ever stepped forward to claim that they had been his secret lover is as big a mystery as the claim that Dodgson apparently hung out with some of them at a sexually mature age, and under the watchful eyes of their Victorian parents, not to mention those of his own friends and family and neighbours, to spend the night under his roof. But perhaps it is true what Leach suggests, that everyone involved had their reasons for keeping silent on

so sensitive a matter and preferred to be enveloped by the mythology built around this towering public figure, rather than spoil the carefully crafted image.

27 The *Velociman* was a tricycle without pneumatic tyres. Dodgson supplied ideas to make it easier to steer and more comfortable to ride [54].

28 To be accurate, I must add that Dodgson lost his right front tooth during the final year of his life, but that does not negatively affect the magnitude of this accomplishment. According to his former child friend, Isa Bowman, Dodgson was in fact so obsessed with his teeth that he saw his dentist on an almost daily basis. As she wrote in her biography of him, *'He had great ideas upon the importance of a regular and almost daily visit to the dentist. He himself went to a dentist as he would have gone to a hairdresser's, and he insisted that all the little girls he knew should go too'* [56]. Ms. Bowman probably exaggerated, as Dodgson must hardly have been able to find the time to see his dentist so frequently. Moreover, during the early 1880s, he had to have three teeth stopped in a single week, and on another occasion had to visit his dentist on eight consecutive working days to have *'a quantity of stopping'*, something that would never have occurred if he had been seeing his dentist on a regular basis. In all, his alleged obsession with daily visits, if true, was probably of a limited duration.

29 Alice Liddell's sister, Edith (1854–1876), for example, died from peritonitis at the age of 22.

30 Dodgson himself indicated that one of his sisters did not stammer, two stammered very slightly, two a moderate amount, and two rather badly [48].

31 The *Ammoniaphone* was also advertised as being effective against *'coughs, colds, clerical throat, bronchitis, asthma, consumption, aphonia or loss of voice, deafness resulting from colds, all affections of the throat and chest, and sleeplessness'*. According to the advertisement, it was constructed of *'a specially prepared, non-corrosive metal, with handles, ebony polished, having patent spring valves'* and *'charged with a chemical compound, combined so as to resemble in effect that is produced by the soft balmy air of the Italian peninsula when inhaled into the lungs, hence the term'*.

32 *Bordetella pertussis* was identified in 1906 by the Belgian microbiologist Jules Bordet (1870–1961)—after whom the microorganism was named—and his colleague Octave Gengou (1875–1957). Shortly afterwards the pair also developed a vaccine.

33 The term *'ague'*, mentioned so often by Dodgson when he was ill, deserves special attention. It is difficult to grasp, as there is no equivalent for it in contemporary medicine. Historically, ague is a

17th-Century notion associated with malaria . Of note, the term stems from an epoch long before the identification of the *Plasmodium* parasite that causes malaria as we know it today, that is, a life-threatening, mosquito-borne infectious disease that is characterised by high fevers and shaking chills. Instead, ague was associated with the bad air (from the Mediaeval Italian expression *mala aria*) that was found in marshlands and other water-rich parts of Europe [66]. Initially the air itself was considered unhealthy and people therefore spoke, for instance, of ‘the malarial nature of fogs’. Subsequent authors assumed that the air contained infectious agents which spread disease, thus conceptualising the air as a *carrier* of disease rather than a pathogen in and of itself. In Dodgson’s time, malaria was still a mysterious feverish disease. By using the term ‘ague’ it seems he had rather meant a combination of fever, tiredness, headache , and vomiting, with or without a fluctuating pattern; or, more loosely, a feeling of general malaise. At any rate, he did not use ‘ague’ as a synonym for ‘a cold’, as he once remarked that he had ‘*a feverish cold, of the bronchial type, and the risk of ague (a form my colds usually take)*’ [67]. Moreover, he mentioned that ague was ‘*said to be chiefly caused by bad drainage*’, adding in a letter to Mrs. Dyer that he would like to visit her again next summer, but that he thought of first sending an expert down to open up the ground in front of the house to examine the connection with the main sewer, and have the premises thus certified to be safe [68].

34 I was unable to retrieve what ‘black dose’ used to be (the third medicine prescribed by Dr. Hussey), so this does not provide any further clues as to what Dodgson had actually been suffering from.

35 Because of the combination with lumbago, or backache, Goodacre suggests that Dodgson may have suffered from a complication of cystitis called pyelonephritis, an infection of the kidney pelvis [24].

36 In September 1871, mere weeks before Dodgson had himself vaccinated for the second time, the *Sunderland Times* reported 120 cases of smallpox during the previous month in the Gateshead Union, of whom seven had died in the fortnight [98].

37 It was fortunate, that in Dodgson’s time a vaccine against smallpox was available. This was thanks to the work of Dr. Edward Jenner (1749–1823), a British physician who had discovered that milkmaids who had previously been infected with cowpox never went on to develop smallpox. This led to the first variolation programmes, which were followed by vaccination programmes such as those employed later by the WHO . Today no-one is vaccinated against smallpox, although governments keep large quantities of vaccine in store to be distributed nationwide in case of a new outbreak, whether or not in the context of biological warfare.

38 Incidentally, sulphurous acid is not to be confused with *sulphuric acid* , a corrosive mineral acid that, undiluted, is bound to cause serious burns.

39 *Rubia tinctorum* is an herbaceous perennial plant, also known as common madder or dyer's madder. Its root has traditionally been used to treat 'obstruction of the spleen', and as a homeopathic solution it has been recommended for '*anaemia and under-nourished conditions, especially in splenic anaemia*' [107]. However, there is no scientific evidence that *Rubia tinctorum*, in either form, has any beneficial effects on the spleen. Besides, it is entirely unclear on what grounds something was considered wrong with Dodgson's spleen.

40 The term *fortification of Vauban* refers to the French engineer Sébastien le Prestre Vauban (1633–1707), who designed a zigzag type of fortification wall that was most effective against enemy attacks.

41 In this context, it is also worth noting that Podoll and Robinson drew attention to the split-body image of Sylvie in Dodgson's later book *Sylvie and Bruno*, and speculated that in this case, too, he may have used a migraine -associated illusion, experienced by himself, as a source of inspiration [138].

5. Neurobiology

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

The symptoms characteristic of Alice in Wonderland syndrome are usually subtle in nature, in the sense that they tend to affect only a minor aspect of a person's full perceptual experience. As a consequence, everything is perceived just as before, except that, for example, all vertical lines are slanted, as happened to Paul; or time is found to slow down, the way this is experienced by Neo in *The Matrix* [1]; or one's body is experienced as shrinking to the height of a thimble, as happened to Alice in the story and to Ms. Rembrandt in real life and so on, in accordance with any of the numerous variations that we have encountered so far, and indeed many, many more. Sometimes these symptoms occur in conjunction with each other, although they mostly present as isolated perceptual distortions, leaving the rest of what we perceive intact. What is more, even when several symptoms are present, they tend to be experienced in the same sensory modality, being all visual in nature, or all somatosensory and so on. As far as we know, based on the limited number of extant case descriptions, only 15% of all people with Alice in Wonderland syndrome report symptoms in more than one sensory modality [2]. The remaining 85% of them report unimodal experiences—which, as we just saw, tend to consist of a single symptom.

To understand how it is possible that only a single aspect of our perceptual experience can be altered, whereas the rest remains the way it had always been, we need to turn to neurobiology and realise that the

perceptual system is not like a video camera (that offers a series of snapshots representing scenes in front of the lens) or a microphone (that converts vibrations in the air into electrical signals) or even a computer (which merely stores, retrieves and manipulates data). Instead, it is a vast neural network with numerous larger and smaller circuits, connected in a hierarchical manner, which together process perceptual information at numerous disparate locations and in numerous ingenious ways. Higher cortical circuits ensure that, in the end, all that information is reassembled in such a way that the resulting percept is coherent and complete, to the extent that, in visual perception, even the blind spot is ‘smoothed over’ and, in audition (hearing or listening), words are ‘filled in’ when our reception of them is hazy. Moreover, to produce such a complete and coherent whole, these higher cortical circuits are dependent on the workings of lower-level circuits, all the way down to tiny stacks or clusters of individual nerve cells called *cortical columns*. It is said that ‘the devil is in the details’—and that certainly holds true for Alice in Wonderland syndrome.

Ironically, perhaps, initial research on those tiny cortical columns, which are located in a part of the brain called striate cortex, was carried out at almost the exact time that Todd was busy designing his syndrome of Alice in Wonderland. During the 1950s, unbeknownst to each other, Todd was working away on his clinical syndrome at Littlemore, while two scientists on the other side of the Atlantic Ocean conducted basic research on the working of the cat’s striate cortex. These two men were David Hubel (1926–2013) and Torsten Wiesel (b. 1924), who, in 1981, went on to receive a shared Nobel Prize in Physiology or Medicine for their groundbreaking contributions to unravelling the neural machinery that underlies visual perception. Higher circuits of the perceptual system may be able to iron out some of the system’s inherent ‘flaws’ by attaching meaning to incomplete data (as with the eye’s ‘blind spot’) and by suppressing information that is deemed irrelevant (as in the case of afterimages and floaters)—however, in the end, they have to deal with the raw material supplied by lower-level circuits, such as those conceptualised by Hubel and Wiesel. In accordance with the principle of ‘garbage in, garbage out’, the raw material affects our perceptual experience as a whole and, if corrupted, alters it in ways that higher cortical circuits are unable to correct, no matter how hard they try to ‘smooth things over’ or ‘fill in the gaps’.

In all likelihood, Todd and his illustrious American colleagues never met and were, moreover, oblivious to each other's work. That is probably the main reason why their respective contributions to our understanding of visual perception developed along strictly separated lines. When I emailed the only surviving member of this trio in January 2018, Professor Torsten Wiesel was approaching the respectable age of 94 years. Nonetheless, he was kind enough to email me from his account at the *Karolinska Institutet* in Stockholm, Sweden, where he had begun his career as a medical student in 1947 and (having spent most of his working life in the USA) still held a part-time position in 2018. He confirmed that he had never heard of John Todd, nor of Alice in Wonderland syndrome. After looking at the paper I had sent him, he wrote

My thanks for your most informative message and attachment. I did not know or at least don't remember anything about the syndrome so well described in the attachment. Even though I grew up in a mental hospital where my father was director and did some psychiatry as a young doctor, I have unfortunately in my old age (94 years in a few months) lost my interest in trying to understand or speculate about the possible neural basis of various mental states.

That Professor Wiesel, at his advanced age, no longer invested his precious time in trying to fathom the neural basis of perceptual distortions was, of course, totally understandable. Nevertheless, I could not help but ask myself what unforeseen directions his work might have taken if, during his active years, he had been aware of Todd's approach to psychopathology and of the possibilities it offered for a clinical application of his own celebrated work on cortical columns. In what follows, I therefore seek to incorporate Torsten Wiesel's work—and that of his long-standing scientific partner, David Hubel—in what we already know about Alice in Wonderland syndrome. So here we go, examining a line of research carried out during the 1950s, to see how it can help us understand another line of research from the 1950s, which has only recently started to move into the spotlight.

5.1 Visual Distortions

The visual distortions described by Todd in 1955—that is, metamorphopsias—have since been reported in around 90% of all published case descriptions on Alice in Wonderland syndrome [2]. Even in the absence of prevalence rates from systematic, large-scale studies—which simply have not been carried out—these figures accord with my own clinical impression that the visual modality is indeed the one that is most frequently affected. It is therefore tempting to believe that metamorphopsias are also the most important symptoms of Alice in Wonderland syndrome, although, obviously, we are currently not in a position to say that. What we do know is that metamorphopsias present in many shapes and varieties. In the preceding chapters, we already met several persons suffering from widely varying types of metamorphopsia. For those who wish to see what else may go wrong in the visual modality when a person suffers from Alice in Wonderland syndrome, Table A.2 (Appendix A) provides an overview of all the metamorphopsias described in the medical literature. Although it lists more than 40 types, even that collection is not exhaustive. For instance, at my outpatient clinic in The Hague, I sometimes hear people describe the corners of a room being torn open, like those of a worn-out shoebox, and its contents being sucked through the cracks, leaving them behind in an empty room. As far as I know, there is no literature on this peculiar visual phenomenon and, to be honest, I have no clue as to how it should be called, let alone how it should be explained in terms of underlying neurobiological processes.

Fortunately, other metamorphopsias are straightforward in their presentation—even though they may puzzle those who experience them, they can often be easily connected with parts of the visual network responsible for their mediation. In many cases, these are circuits—or relatively small components of circuits—at the lower levels of the perceptual system's hierarchical organisation. To get an idea of how these lower-level functional units affect our visual perception as a whole, let us recall our high-school biology lessons: we were taught that all visual perception starts out with light rays (or photons) falling on an object and being reflected off its surface, to then enter the eye and pass through the cornea and lens, which together converge those light rays in such a way that they project in an inverted fashion on the retina. It is well known that the retina is a light-sensitive membrane at the back of the eye, which contains rods and cones that react to the incoming photons by producing neural

impulses, or weak electrical currents. From both eyes, these neural impulses are relayed to the optical nerves (which lead straight into the brain) and meet each other at the optic chiasm, a neural intersection within the brain itself, which makes half of the nerve fibres from each eye cross over in such a way that information from the left part of the visual field of both eyes ends up at the right side of the brain and vice versa; all this happens in a part of the brain (located way back in our head) that we know as ‘visual cortex’. When that part is activated, or so we learned a long time ago, we *see* the object in front of us. As we realised back then, we do not actually ‘see’ with our eyes but with our brain—more specifically, with that part of the brain that is removed as far away as possible from our eyes. I am certain that no engineer would have designed a brain this way—but, then again, we are light years away from designing anything that comes even close to the human brain.

There is a philosophical side to this account of visual perception (which I shall bypass here) that challenges the notion that neurophysiological activity in visual cortex can indeed be equated to ‘seeing’. Instead, proponents of this critical tradition tend to offer a dualistic model that designates ‘seeing’ as an emergent mental state of brain activity—or as a parallel state, for that matter—but, at any rate, debate the notion that neurons firing in visual cortex (‘matter’) can be equated with the event we call seeing (a ‘mental state’). Irrespective of whether or not we should consider that line of thought worth pursuing, there is another problem with the traditional account of visual perception that requires our attention, that is, the somewhat simplistic way in which it describes the route taken by visual information from the eyes towards those areas that make us aware of its contents (whether or not through that extra little hop into the ‘mind’).

The part of the perceptual network traditionally endowed with making us aware of complex visual scenes is called *visual association cortex*. However, what we never learned in high school is that visual association cortex depends on numerous signals from the visual network as a whole, including signals from specialised groups of neurons, which are thought to be similar to the cortical columns described by Hubel and Wiesel on the basis of their famous animal experiments.

Incidentally, the notion of cortical columns was not Hubel and Wiesel’s own invention, but was introduced by another American neuroscientist, Vernon Mountcastle (1918–2015). Upon hearing about his work, Hubel and

Wiesel decided to search for similar microstructures in a part of the brain that hardly anyone seemed interested in at the time, that is, visual cortex . They did that by making single-cell recordings of neurons inside striate cortex of cats. It was a purely experimental endeavour, initiated by them at Johns Hopkins University in 1958 and continued for many years at Harvard, where the two of them explored these parts of the cat brain much in the way seamen in Columbus ' time crossed the ocean—without a clear idea as to where they were headed. The bodies of the cells they studied, all the way in the back of the cat's brain, had a diameter of 4–100 μm , which is one four-thousandth to a tenth of a millimetre, which is almost too small to picture, especially if you use an ordinary ruler for reference. In their attempts to activate those tiny cells, they first taught themselves the dazzling technique of inserting microelectrodes into the brains of live cats. They did that by drilling a 2–3-mm hole through the skull and the underlying *dura mater* (the thick membrane enveloping the brains of mammals) and then sticking a small tube through it. After waxing the space between skull and tube, they then inserted a tungsten electrode through the tube and manipulated it (without anything as fancy as X-ray guidance) into the cat's striate cortex. If that was not challenging enough, they subsequently started shining tiny beams of light into the cat's eyes, thus projecting circular spots of light onto its retina, as well as black spots against a light background, red lines through a slit, and so on in numerous variations, hoping that one of those light stimuli would call forth a response from that single, tiny cell connected to their microelectrode.

Needless to say, with this set-up, they had given themselves an almost impossible task. After all, which part of the retina should they stimulate? And with what kind of light pattern? And how could they expect that any of those stimuli would activate that particular cell connected to their tungsten microelectrode, all the way in the back of the cat's head? It was not like they attempted to record the reaction of visual cortex as a whole—rather, they focussed on one in several millions of cells that could theoretically be activated. In other words, it was worse than looking for the proverbial needle in a haystack—which is to say nothing about handling those cats in such a way that would make the whole experiment possible in the first place. As the two pioneers commented afterwards, this was '*before the animal rights groups had made it so much harder doing research involving animals*' [3]—so perhaps it is best not to ask any further about the minutiae

of their experimental procedures. At any rate, they tried and tried and, after numerous failures, to their surprise they found that one particular cell, which they had stubbornly held on to for a while, did indeed start to respond, albeit to a different light stimulus than the one they had projected onto the cat's retina. As they recounted afterwards,

The break came one long day in which we held onto one cell for hour after hour. To find a region of retina from which our spots gave any hint of responses took many hours, but we finally found a place that gave vague hints of responses. We worked away, in shifts. Suddenly, just as we inserted one of our glass slides into the ophthalmoscope, the cell seemed to come to life and began to fire impulses like a machine gun. It took a while to discover that the firing had nothing to do with the small opaque spot—the cell was responding to the fine moving shadow cast by the edge of the glass slide as we inserted it into the slot. It took still more time and groping around to discover that the cell gave responses only when the faint line was swept slowly forward in a certain range of orientations. Even changing the stimulus orientation by a few degrees made the responses much weaker, and an orientation at right angles to the optimum produced no responses at all [3].

That is how the two of them got a single cortical neuron to respond to the cat's perception of lines at a vertical orientation. It was a major accomplishment, and a major discovery, which formed the basis for their now classic paper, published in 1959 in the *Journal of Physiology* [4]. In it, Hubel and Wiesel described what they called 'orientation columns'. Like Mountcastle before them, they thought of these tiny units as vertically organised stacks of cortical neurons—literally stacks of nerve cells that reacted by turning either 'on' or 'off' in response to visual stimuli of lines under a certain angle. After this first cortical column, they described similar ones that responded to black lines but not to white ones and those that did the reverse or responded to both; ones that responded to red lines but not to black or white ones; ones involved with stereo vision, and so on, detailed, in the flurry of papers that followed, as a seemingly endless variety of tiny functional units of visual information processing. We now also know of cortical columns specialised for colour, movement direction,

spatial frequency and myriad other variables. Contrary to what was initially thought, many of these tiny functional units turned out to react to more than one particular stimulus. Moreover, many of them do not appear to consist of literal stacks of cells, but rather of clusters of cells without any well-circumscribed borders. And yet the central notion of very small, yet highly specialised functional units has remained, along with the notion that these are found at the lower levels of the hierarchically organised visual network [5] .

In the intervening decades, studies have indicated that such tiny units also exist in human visual cortex [6]. The reason why neuroscientists are still not fully certain about their exact nature and even doubt whether, in humans, they have the shape of vertical stacks of cells (as proposed by Hubel and Wiesel) is that post-mortem studies of the human brain consistently fail to show any anatomically segregated cellular columns with a vertical orientation, and thus fail to provide visual conformation for their existence. As a consequence, we have had to rely on the study of live human brains to find out whether they exist. To do so, neuroscientists have projected light stimuli with lines and gratings onto the retinas of test persons and sought to measure neural activity in the backs of their heads. For obvious reasons, they did not do so by inserting tungsten electrodes into the brains of their test persons, the way Hubel and Wiesel had done with their cats, but rather by inviting the participants to take place inside an MRI scanner and then measuring changes in the activity of their brains concomitant with the visual stimuli projected onto their retinas. Even though the spatial resolution of today's MRI scanners is too coarse to get functional units of such a minute size to register individually, under these circumstances the brain does respond selectively to retinal stimulation with lines and gratings of different orientations. What is more, functional MRI studies indicate that small-scale units that respond to lines of different angles lie intermingled in striate cortex , just the way Hubel and Wiesel had reported in cats and other mammals [7]. That is how we know that their findings are as relevant for human visual perception and that (even though we may still have to establish the exact details of the way things work in humans) we, too, depend on the proper functioning of numerous discrete cortical neuron populations for the processing of visual stimuli.

Thus, because of the legacy of Hubel and Wiesel, and the tentative confirmation of their findings by imaging studies in humans, I was able to

tell Paul and his mother that the reason why he saw everything as slanted was probably that something was wrong with his orientation columns. More specifically, my guess was that, in his case, the orientation columns for vertical lines had ceased to switch on when they should, and that, in their stead, those for oblique lines had started to switch on, making him see all vertical lines as slanted. Because the size of cortical columns prevents them from showing up individually on MRI scans and, moreover, any focal electrophysiological activity that might deprive them of their function is too subtle to register on an EEG, it was no wonder that these auxiliary investigations had shown nothing out of the ordinary in Paul's case. Nevertheless, my guess was that the orientation columns for oblique lines had taken over the function of those for vertical lines, probably because they were no longer switched off by lateral inhibition. Lateral inhibition is a process by which neighbouring neuron populations with different functions cancel out their next-door neighbours when they themselves are activated. This is a tried and tested mechanism in brains throughout evolution, thus allowing for maximum signal strength and minimal interference. In Paul's case, the orientation columns representing vertical lines seemed to have lost their ability to switch on and, thereby, also their ability to suppress the spurious turning-on of adjacent columns, which represented oblique lines.

Incidentally, orientation columns are not the only functional units to be found at this very low level of organisation of the brain's visual system. Some of the cortical columns described by Hubel and Wiesel have a function in stereo vision, which is a prerequisite for depth vision. As you will remember, Alice, at some point, encounters the gardeners with their flat, oblong appearance, to be joined later by the soldiers, and the King and Queen of Hearts, which are all depicted as live playing cards. When in real life we see all things as flat, the way Alice perceived these characters, we speak of *loss of stereoscopic vision*, a symptom we encountered before. This type of metamorphopsia can be caused not only by the loss of function of one eye, but also by the brain itself, when it ceases to create the illusion of three-dimensionality, a function that we are so accustomed to that we normally do not even think about it. The cortical columns responsible for this are the ones devoted to stereopsis, located in striate cortex, or V1, along with cells with a similar function in area V3/V3A, identified by Hubel and Wiesel in their later work with monkeys, and, from then on, referred to as *binocular depth cells* [8].

However, the human visual system is not merely a collection of tiny stacks of neurons switching on or off in response to specific input signals. While such tiny ‘stacks’ (or whatever their actual shape may be in humans) are certainly part of it, and an important part for that matter, we should not forget that the system as a whole is a hierarchical network and that, ‘higher up’ in the network, much larger neuron populations are active—some of which, however, also have specialised functions. An example is visual area 4, or V4 for short, a relatively large structure located in the ventral occipital lobe, which used to be called the brain’s ‘colour centre’. Even though we now know that other cortical areas also play a role in the processing of colour (ranging from V1 to V3) area V4 nonetheless plays a major role in it. Philosophers have asked themselves for millennia whether colours exist ‘out there’ in the world where we see them, or whether they might perhaps be created by ourselves. When we look at a field full of bright red poppies, bathing in the sunlight, is the colour red inherently present in their petals, or do those petals somehow *trigger* us to see the colour red? Plato (c. 427–347 BC), and even the Presocratics (none of whom had any notion of the workings of the brain, let alone of the visual network), had elaborate theories on how colour is created by ‘*the fire emanating from our eyes*’ and ‘*the fire emanating from objects*’, thus already debating the naïve point of view that colours simply exist ‘out there’ in the outside world, for us to be registered by merely looking at them [9]. The interest of these ancient philosophers in the origin of colours may well have been fuelled by people with colour blindness, who may fail to distinguish between red and green, for example—or by the writings of Homer, whose description of the sea as ‘*wine-dark*’ and other peculiar remarks on colour have led historians to believe that he may have been colour-blind as well [10]. During the 17th Century, the debate on colour was given a facelift by posing the question whether colours are ‘*primary*’ or ‘*secondary*’ qualities of objects, that is, whether they are inherently present in objects themselves, or whether they are *produced* by those objects in an observer [11]. During the 19th Century, Nietzsche, in his characteristically bold style, took that question to a whole new level by offering,

We observe all things through our human head, and cannot cut that head off; and yet the question remains what would be left of the world if you cut it off anyway [12].

It is not difficult to see what that question implies for colours: Would we still be seeing colours if we were able to look at the world without having to use our head and the perceptual system contained therein? The question is unanswerable, of course, since we cannot perceive anything without engaging the perceptual system. But at least we now know that, without area V4, we would only be seeing things in black and white, whether or not colours actually exist 'out there'.¹ What the philosopher with a hammer must have suspected, well over a century ago, is something that empirical science has taught us in the meantime, namely, that the subjective experience of colour does indeed depend on the brain. Without V4, or with V4 being temporarily switched off, we suffer from *achromatopsia*, a type of metamorphopsia characterised by a lack of colour [13].

Being much larger than the orientation column s, and having a place much higher up in the visual network, V4 not only subserves the perception of colour but also plays a role in the processing of shape, texture, brightness, orientation, curvature, stereopsis and motion. Regarding motion, however, another part of the brain is of even greater importance. That part is the middle temporal visual area, also known as V5, another relatively large neuron population, located in extrastriate cortex. You may think that the seeing of movement is simply a matter of looking at some object moving through your visual field; however, we need V5 to *actively add* the factor of movement for us; otherwise, we are incapable of visually perceiving it. That may sound rather abstract—but let us see what happens when this mysterious structure stops doing what it normally does so effortlessly that you and I do not even think about it when we go about our lives.

Some time ago, a male in his 50s, Mr. Janssen, had been referred to me because he had been inactive for some 30 years. Not just inactive in the sense of hanging around with friends in bars or occupying himself with hobbies and chores while he should have been pursuing a career but really, almost totally inactive. This is something we see occasionally in people suffering from chronic psychosis; one can imagine that it may also affect those suffering from chronic depression or untreated catatonia, although, even in those cases, 30 years would be an unusually long period of time for someone to be that inactive. Because of his extreme lethargy, for more than half of his life, Mr. Janssen had been treated on-and-off with antidepressants, antipsychotics and numerous other types of medication, without any result other than that he became even more lethargic due to the

side effects. The physician who referred him to me had taken over duties from his predecessor and had been very keen to distil out of the man's story of chronic inactivity that there was something wrong with his visual perception. However, he was unable to tell what it was—and neither could Mr. Janssen himself. However, this new physician insisted that I should see him for a consultation, in the hope that I would be able to pinpoint what had been wrong with him all those years.

When I saw Mr. Janssen, together with one of my residents, Maaïke van Gent, we made the acquaintance of a remarkably lively man, a bit short of stature, but looking quite youthful in his sport-lifestyle clothing, who did not strike us at all as someone suffering from chronic psychosis or depression. Nevertheless, he confirmed that he had not been able to do much more than lie on the couch ever since his early twenties. He used no substances worth mentioning and said that he would certainly like to work if he could, but that he could not. When we asked him about his visual perception and showed him the PowerPoint presentation with images of metamorphopsias, he recognised none of them, except for the one that shows a ballerina with a series of copies of herself trailing behind her, photographed with the aid of a stroboscope. What he recognised specifically was how she was depicted as if moving by leaps. As Maaïke guessed correctly (and you may also have guessed by now), it turned out that he had suffered for the past 30 years from *akinetopsia*, a very rare type of metamorphopsia, characterised by the inability to perceive movement [14]. Thus, instead of seeing people walk from A to B, Mr. Janssen saw them popping up in different places while they passed through his visual field, as if they temporarily ceased to exist and were then recreated a fraction of a second later in a different location; you will get the idea if you have ever seen people dancing in a disco under stroboscopic light. It might seem a minor inconvenience in comparison with the hearing of derogatory voices, or with the debilitating effects of a major depression, but in everyday life we are so dependent on V5 and its function in the perception of movement (whether it be in traffic or in the relatively safe environment of our homes), that it is indeed almost impossible for us to function properly without it.² Come to think of it, we cannot even watch TV when we have this condition—which even the demented elderly can still do.

To further illustrate what V5 does, let us have a look at the logical opposite of *akinetopsia*, a condition called *Riddoch's phenomenon*.

Riddoch's phenomenon is characterised by a combination of cortical blindness and an *intact* ability to see movement. This curious phenomenon was first described in 1917 by George Riddoch (1888–1947), a Scottish neurologist. Before becoming a neurologist, Riddoch had served during the Great War as a temporary captain in the Royal Army Medical Corps, in which capacity he had come into contact with soldiers who had been shot in the back of the head and had miraculously survived that, even though some of them had lost their entire visual cortex. This had rendered them cortically blind, meaning that they were unable to see anything, even though their eyes were still functioning properly, and the neural impulses generated by their retinas were still being relayed all the way to the back of their head—where, however, they encountered a gaping hole where visual cortex should have been. What Riddoch discovered, was that some of these cortically blind soldiers were indeed blind to light, colours and shapes, but not to movement. It may be hard to imagine, but what these unfortunate soldiers saw was pure movement—without any objects or scenes *being* moved. As recounted by those who had fought side-by-side with these cortically blind men, allegedly some of them had still been able to aim correctly as soon as enemy soldiers had come storming in the direction of their trenches. This only makes sense if we realise that V5 is not located right at the very back of the head, but slightly more to the front and that it may, thus, stay intact when visual cortex is destroyed.

Over the years, many people have told me that they see stationary objects moving in their direction (or, alternatively, moving away from them) or that they see movement in the walls or in other inert surfaces. In such instances, V5 may have spuriously turned 'on' when it should not have. The same probably holds true for a phenomenon I myself am familiar with, which is the seeing of movement in the corner of an eye. Sometimes, while standing in the kitchen or sitting on the couch, I have the sensation that one of our cats is moving through the periphery of my field of vision. However, when I turn to look, nine times out of ten, there is no cat. When it happens again, and I focus on what I actually see, I always realise that all I perceive is movement. I merely *think* of a cat because they walk through our house all the time, but that is not what I see in such instances. The reason for this to happen is spurious activity in the rods, the photoreceptors located in the periphery of our retina, which may become so sensitive to

movement—especially after 40 years of age—that they sometimes erroneously relay signals to V5 when nothing is actually moving.

As these examples indicate, metamorphopsias are distortions of highly distinct aspects of our visual perception, caused by dysfunctioning of equally distinct constituents of the brain's visual network . However, we need to remember that those structures function in the wider context of the perceptual system as a whole. That certainly holds true for the fusiform gyrus , another bilateral structure, located at the base of the brain, which we already met in the context of Dodgson's inability to distinguish individual faces (i.e. prosopagnosia). The same bilateral structure was the one which we had initially considered to be responsible for generating the dragon faces perceived by Mrs. van Nuys—although our MRI scans did not confirm that. Having an important function in the identification, recognition and representation of faces, in conjunction with the face-representing network as a whole, dysfunctioning of the fusiform gyrus may alter our perception of faces in such a way that single aspects (such as eyebrows, eyes or mouths) are grotesquely deformed in each and every face that we perceive or, alternatively, in our own face when we see it in the mirror . The latter type of prosopometamorphopsia , as this type of visual distortion is called, was what Willem suffered from. Looking in the mirror , he sometimes saw his face being indented by a brick-like shape, with one eye protruding over the edge of its socket and gradually slithering down his cheek. As he never observed this bizarre deformation in other people, we may conclude that the perceptual system apparently makes use of different mechanisms, in all likelihood sustained by different parts of the fusiform gyrus , to represent one's own face versus the faces of other people. As we saw, Ms. Rembrandt had similar experiences, perceiving her own face in the mirror as large and bloated and furrowed, whereas other people's faces remained unaltered. Mrs. van Nuys, on the other hand, used to see *other* people's faces take on the likeness of a dragon. What made her case so special, and indeed so rare, is that the deformation was not limited to a single facial feature such as an eye or a nose, but that all aspects of people's faces were thus replaced by the facial features of dragons.

These examples confirm that human visual perception does indeed depend on the orchestrated action of numerous smaller and larger neuron populations with specialised functions for the encoding of different aspects of our visual input. Some of those functions involve the representation of

the tiniest imaginable units of visual perception, such as lines of a certain orientation, or light–dark contrasts, whereas others involve extremely complex configurations such as human faces. In order to render an accurate representation of scenes in the outside world, the higher cortical centres of the brain need to be able to rely on the input from all those numerous units to piece those scenes together. The examples given above may serve to give us an impression of what may go wrong when those units at the lower (and sometimes higher) levels of organisation fail to do what they should do and—while we are at it—may fill us with awe that things do not go wrong more often in our everyday lives.

A final point that I wish to touch upon for now, is something that was hinted at in the story of Mrs. van Nuys, but not yet fully elucidated. As we saw, the transformation of human faces into dragon faces that she experienced was always preceded by a symptom-free interval of one to several minutes. Such a latency period is in fact reported by many people suffering from visual distortions. It is such a striking and consistent feature that even the early pioneers in the area of visual perception did not fail to notice it [15–17]. Speculating about its cause, they came up with the name *cerebral asthenopia* to convey the idea of a heightened fatigability of otherwise normally functioning neuron populations. Since, in such cases, the perceptual system is apparently capable of representing faces or other visual input undistortedly (for however brief an interval), the neural mechanism underlying it must be structurally intact. The fact that after several seconds or minutes these flawless representations give way to distorted ones, means that these neuron populations cease to exert their normal function. Given that we do not exactly know what causes this to happen, ‘heightened fatigability’ seems a pretty good name to cover the neural mechanism that leads up to metamorphopsias. Since no-one knows which biochemical process (or processes) may underlie that central mechanism, I think this is a topic that certainly needs exploring in future studies.

5.2 Distortions in Other Sensory Modalities

Although other types of distortion appear to be considerably rarer, they are just as spectacular as the visual symptoms of Alice in Wonderland syndrome. Moreover, the way they are mediated is at least as fascinating.

To get an idea of how it is possible that people may sometimes experience their body as becoming larger or smaller than it is, or see their hands change into witches' claws, or suddenly have the sensation that their skull is open to the air, allowing the wind to sweep over their unprotected brain (as one of my patients once described to me in great colour and detail), we need to realise that we are never in direct contact with our body.

Yes, you just read that: we are never in direct contact with our body.

You may think that you know your body pretty well, but you don't.

How could that be? After all, we look at ourselves numerous times per day, glancing in the mirror in the bathroom, in the hallway, in the dressing room, in our car, at school, at work; moreover, I am not even talking about all those other places where we see ourselves visually represented nowadays: in the reflecting surfaces of shop windows, in photographs, on film, on the closed-circuit TVs of surveillance cameras, in selfies on our phone and so on, in numerous places, perhaps even more than many of us feel comfortable with. And yet all that imagery does not bring us into direct contact with our bodies. Not even when we use the simplest device of all, the mirror.

Mirrors go a long way back, to 8000 years ago, when the first ones were probably made in what is now called Turkey. They were genuine works of art, shiny and slightly convex, made out of a volcanic glass called obsidian [18]. Obviously, the common people had no idea that they even existed. The few mirrors that were so skilfully manufactured were locked away inside palaces, to be used only by Emperors and Sultans, and to be showcased by them in front of envious guests. Long after that came mirrors of polished bronze and copper, whereas metal-coated glass mirrors have only been available since somewhere between the first and 3rd Century AD—initially, again, only to the extremely wealthy, but today, obviously, to anyone with a bit of money or the resourcefulness to pick one out of a dumpster. For us, today, surrounded as we are by mirrors and other reflecting surfaces, it is astonishing to realise that for millennia, the majority of people hardly ever got to see their own face except, perhaps, when they occasionally looked down into a pool of water or found some other shiny surface that might reflect their features, such as a knife or a polished piece of marble; even then, they could hardly have been expected to get a proper, undistorted look. Since nowadays most of us cannot manage to get through a single day successfully *avoiding* a representation of our own face, we tend to think that

we have a fairly accurate idea of what we look like. However, as I said, looking at ourselves in a mirror does not bring us into direct contact with our bodies. In fact, the story about Willem, who sometimes saw his face as if bashed in by a brick, should have already made us suspicious of what we ourselves are looking at when we look into a mirror. If there was such a huge difference between the face that Willem saw and the one he felt with his fingertips, what exactly did he look at—and what exactly do we all look at, when we look into a mirror ?

It may sound obvious, but when we look into a mirror, all we see is light reflected back at us, giving us an impression of colours and shapes and movement. As we know by now, we do not even see all that with our eyes. Our eyes help us to converge the light rays onto the retina and to thus generate neural impulses that travel all the way to the back of our brain. Eventually, it is the brain that *actively creates* an image of what our body is supposed to look like. Even though it does so on the basis of the light reflected back to us through the mirror, it *creates* that image on its own, the way it creates images of everything that we observe. Since we already know that the visual system is not like a video camera, and that it has quite a few degrees of freedom, you will realise that it does not necessarily represent our bodily features one-on-one.

Knowing that, you will probably also realise why so many people have difficulty, when merely looking into a mirror, in assessing whether they have gained or lost some weight. I myself love my daily rounds through the park in our neighbourhood—nothing spectacular, just a bit of cardio to stay reasonably in shape—and, having done so steadfastly for several years, I must have lost some 20 pounds in the process. Nevertheless, when I look into the mirror, I do not really have the impression that I am any leaner than before. It is only when I meet people who have not seen me for a while, that I hear them say, ‘*Hey, you look so much thinner ... have you been running or something?*’

So, our brains create a visual image of what our bodies look like. But then again, we also *feel* a certain way about our bodies, don’t we? We can feel whether we are fat or not—by touching our belly with our hands, for example, and squeezing those tender love handles between our fingertips—but even without such tactile information, your body just *feels* a certain way, from the inside, on the basis of which you probably have a fairly accurate sense of how tall you are, how fat you are, and what your body

must be like on the whole. That was probably how people—before mirrors became a household product—nevertheless had some idea of what they looked like. However, similar to the way the visual system conjures up a body image that is not necessarily reliable, it does something similar to bodily sensations. On the basis of numerous impulses from within the body, and from those that interact with any of its outer surfaces, it creates a representation of the body, which is called the ‘body image’. The body image (also referred to as body schema and *image de soi* in the older literature) is an active product of the brain, which determines how we experience our bodies. When we direct our attention at our bodies, what we get, instead, is the latest version of our body image; however, this image may not always easily catch up with changes to our actual body and may, occasionally, deceive us completely.

We have all heard about people who lost a limb in an accident and, subsequently, went on to experience their missing arm or leg as if it were still there: the so-called ‘phantom limb’—simply being there or hurting or itching or conveying the sensation that it is bent or twisted in an uncomfortable position [19]. Apparently, even a dramatic change such as the loss of a limb may give the body image a hard time catching up. However, when this system goes truly off the rails, it can do so spectacularly. We already know how dysfunctioning of cortical columns (and sometimes larger neuron populations) in visual cortex can change the world-as-we-see-it, subtly yet profoundly. Something similar can happen to the somatosensory system, which is responsible for conjuring up our body image. Did you ever wonder why so many women (along with a few men) who suffer from anorexia nervosa experience themselves as grossly overweight, whereas it is clear for anyone who cares to look beyond the camouflage of their oversized clothing that they are all skin and bones? And what to think of those who suffer from a rare condition called ‘supernumerary phantom limb’, who have the sensation of having an *extra* arm or leg? [20] Obviously, those people are perfectly aware that they were born with just two arms and two legs and could not have grown an extra limb in the meantime; however, they might be so convinced of their predicament that they may plead with a surgeon to remove it—or, more likely, a series of surgeons, since they may be persistent at this, and surgeons do not tend to apply a scalpel to things they cannot see.

I myself was once consulted by a former teacher in his fifties, called Mr. Müller, who had the continuous sensation that the muscles and vertebrae in his back were moving—to such an extent that they seemed to be relocating themselves—and who, moreover, had the recurring sensation of a wing-shaped piece of flesh fluttering on top of his right shoulder. He referred to that piece of flesh, which he could see *and* feel, as ‘*a gill*’ (since it reminded him of the gills of fishes). When he came to see me, he had experienced these highly disturbing sensations for 7 years and had not been able to work ever since the fluttering had started. After I had examined his back, his shoulders and his neck, and had told him that there was nothing out of the ordinary to be seen, I went on to explain that, instead, his body image appeared to be playing tricks with him. I took my time drawing a brain and elaborating on the somatosensory areas and, when I was finally finished, he nodded understandingly. He was a smart man who had lectured in front of highly gifted children, so he had no trouble following my gist. However, since I was not entirely sure that I had convinced him so quickly, I then suggested that we might have a surgeon on duty in our psychiatric hospital, whom we might call and whom we might ask to remove this fluttering ‘gill’ of his. Although I had made it abundantly clear to him that this was only a hypothetical proposal, he immediately and hopefully consented. It pained me to see the spark of hope in his eyes and was also shocked to see how this intelligent man was so utterly convinced of the reality of his condition.

Incidentally, Willem, the student whom we met several times before, also experienced bodily distortions. Once, as he told me, he had seen his left hand grow to about twice its size, whereas the right one had retained its normal proportions. Looking on in amazement at the long, pointy fingers he appeared to have grown on his left hand (with an old, weathered aspect, like that of furrowed branches), he was even more amazed when an additional pair of fingers started to grow out of the same hand, at an angle of 90° to his actual fingers. In Willem’s case, this lasted only a few minutes. Being accustomed to all the perceptual distortions he experienced so often, and having a unique insight into his own situation, he never believed that his hand had actually been deformed this way. And yet that was exactly what his body image told him—and, thus, what he perceived.

Taking things to an entirely different level, people may even experience their bodies to be altered so profoundly, that they are convinced that they

have changed into something different. Thus, they may believe that they are no longer human and have changed into a dog, a wolf or some other animal. The medical literature indicates that this is extremely rare, since no more than 60 cases have been published on this enigmatic condition over the past century and a half; nevertheless, in my psychiatric hospital alone, we identified 8 new cases over the past 6 years [21]. Thus, we had a young man in our hospital who was convinced that his arms had become extremely hairy (which I failed to see), who felt his jaw and teeth becoming larger and harder, and who at night had the sensation that his toenails scratched the bed sheets as if his feet had turned into paws [22]. Being an intelligent, well-educated young man, he had searched the Internet and had come to the conclusion that he suffered from *lycanthropy*, the transformation of a man into a wolf as described in ancient mythology, even though this was at odds with the laws of natural science (or so he said himself). Another young man, admitted to our secluded nursing ward, would howl and bark uncontrollably, sniffle at door posts, urinate in the corner of his room and on the balcony and, when asked about his behaviour, insist that nothing was the matter with him. Only after years of treatment did he confide to one of our nurses that he had been convinced all the time that he was a dog. Similarly, in the nursing home of our hospital, we had a near-blind elderly gentleman who was convinced that his dog was with him, even though this loyal companion of his had died several years before. He would make gestures as if he were stroking the dog and smile when he had the impression that it was licking his hand. However, whenever he became ill due to some recurring infection, he would climb out of bed at night and be found by the nurses, lying on the rug in front of his bed, licking his forearm as if it were a paw, and overall behaving like he himself had turned into his beloved dog. One other patient was convinced that he was a bird, spreading his arms from time to time as if they were wings, and asking disappointedly, upon hearing that the social security services had discontinued his allowance, ‘*Didn’t I lay enough eggs then?*’ Similarly, we had a man admitted who thought he was a cat and hissed at the nurses whenever he did not get what he wanted—as well as several other patients who thought they were wolves or dogs.

As mentioned, the literature suggests that such fundamentally disturbing changes to the body image are extremely rare. However, I find it hard to believe that people suffering from *clinical zoanthropy* (as this condition is

called) would have a preference for converging on the city of The Hague and, for that reason alone, end up in our hospital. As a consequence, my guess would be that the condition is in fact much more common than traditionally assumed, and that there must be numerous other persons out there who experience such profound changes to their body image that even their sense of personal identity is affected by it.

Such cases of clinical zoanthropy—and of phantom limb and anorexia nervosa—lie on a continuum with Alice in Wonderland syndrome. However, we would be stretching the definition of Alice in Wonderland syndrome beyond its rightful limits if we said that they all belong under the same diagnostic heading. I am presenting them here only to illustrate my point that we are not in direct contact with our bodies and that, even though the body image tends to represent the features of our physical bodies reasonably well under normal circumstances, things can go seriously wrong when it stops doing so. Some of the patients whom we have met—Willem, Mr. Müller, Mr. Salvatore, Mrs. van Nuys and Ms. Rembrandt—can certainly testify to this.

Nevertheless, the question remains as to how it is possible that our body image gets so messed up. Since it is shaped by countless impulses from within the body and from the outside world and, therefore, by information about virtually everything that we experience (consciously or not), it is easy to grasp that the underlying system must be a widely disseminated network in our brain—and that it must be larger than the visual network that we examined before. After all, it processes not only visual information but also tactile and somatosensory information and, indeed, information from all our other sensory modalities—shaped, moreover by psychological factors such as memory and expectancy, and even by social factors such as aesthetics and ideas about style and fashion. Essential to integrating all that information are the parietal lobes and, according to several studies, an area called the temporo-parieto-occipital junction, which is the tripoint of association cortex that includes portions of the temporal, parietal and occipital lobes. What exactly goes wrong in that vast system when bodily distortions arise differs between individuals and cannot always be pinpointed in individual cases. However, as we shall see, we do know of numerous conditions that may cause it to go wrong.

However, before we discuss those conditions, let us first examine one more type of non-visual distortion. Although Todd's original paper does

not mention distortions in the sensory modalities for audition, taste or smell, one would expect that these might also be affected in the context of Alice in Wonderland syndrome. And perhaps they are, sometimes, considering Hamed's case description of a man diagnosed with Alice in Wonderland syndrome: this person experienced visual and somesthetic distortions and, in addition, hyper- and hypoacusis, saying about the latter, '*I hear people's voices loud and close or faint and far*'. [23] Since there is hardly any other literature on such instances of auditory distortion whereby voices or other sounds become either muted or heightened (let alone on distortions of smell or taste in the context of Alice in Wonderland syndrome), the topic I would like to address next is the group of time distortions. After that, as promised, we will have a look at the causes underlying all those perceptual distortions.

5.2.1 Time Distortions

The neural mechanisms subserving our experience of time have not been studied as extensively as those underlying somatosensory perception, let alone those underlying visual perception [24]. Nonetheless, we know that the brain and the body as a whole are like a giant clock repair shop, filled with clockworks ticking away at a pace of their own, some of them in synchrony with each other, others at rhythms of their own. Those rhythms are set to numerous internal and external cues, ranging from daylight availability, feeding patterns and changes in oxygen need, to surges of hormone secretion, and the ebb and flow of intracellular calcium levels. In the meantime, many of those rhythms also attune themselves to each other. Although the net result is a unified sense of time, it is as yet unknown whether a separate structure or receptor exists inside the brain that serves as the 'end organ' for all these biological clockworks [25]. However, within the network as a whole, we can certainly point out several processes that are of more importance than others.

The whole idea of time perception stands and falls with the notion that time needs to be cut up into relatively short intervals to enable us to keep track of it. Floating in an isolation tank filled with salt water at body temperature, in the absence of any light or sound or tactile stimuli whatsoever, is almost impossible. What we need is something by which to mark the passage of time. That something is rhythmicity. Some rhythms are given to us physiologically, such as our heart rate, for example, which

provides a natural rhythm that is used by the brain to make estimations of the passage of time. Another (albeit much slower) rhythm is the diurnal pattern of night and day, and an even slower one is the cycle of lunar months. Other rhythms are artificially made by us. Clocks and other chronographs were invented specifically for that purpose, but simply counting may also do the trick, for example, when a musician tries to sustain a chord for a particular duration of time, when dancers dance in silence or when members of a synchronised-swimming team make their moves under water, where they cannot hear the music played above.

Thus, the rhythmicity needed for the creation of psychological time stems from the outside world, as well as from within the body. What is more, it is found at all levels of the body's hierarchical organisation. When we go all the way down to the lowest level of the network for time perception, we find individual genes, in individual body cells, which produce proteins at a fixed rate and, thus, at a rhythm of their own. Since that rhythm differs for different types of cells, if we could make them audible, what we would get from the different tissues and organs made up of those cells would be a cacophony of rhythms. At the highest rung of the brain's network for time perception stands the suprachiasmatic nucleus, a neuron population that is so-called because it is located right above the optic chiasm, the crossroads of the two optic nerves that I mentioned before. Like gene expression at the cellular level, the suprachiasmatic nucleus has a steadfast rhythm of its own. However, being much larger, and being positioned at the top of the chain, it is also much more influential, subjecting numerous lower-level mechanisms throughout the brain, and throughout the body as a whole, to its dominant pace. Because of that, the suprachiasmatic nucleus is also called the 'internal master clock'.

The internal master clock is linked directly to the pineal gland, which secretes the hormone, melatonin, that regulates many phasic processes, including sleep onset, sleep architecture and sleep-wake transitions. The internal master clock also regulates surges in the secretion of other hormones and, moreover, promotes metabolic changes, fluctuations in thermoregulation and changes in autonomic nervous function, which, in turn, regulate processes ranging from activity of the adrenal glands to the secretion of gastric acid. Obviously, all those numerous processes need to be attuned to each other in a coherent way in order to keep the body functioning properly. The internal master clock plays an important role in

accomplishing that. And yet the structure itself is not an atomic clock, ticking away at the same pace whatever the circumstances. Thus, it does take cues from the external world, such as changes in environmental blue light and changes in physical activity, even though it has a strong tendency to stick to its own fixed pace, thus ensuring that behavioural patterns and intrinsic physiological rhythms are optimally synchronised to each other *and* to the 24-hour light-darkness cycle.

When our experience of time is altered, we speak of time distortion s and we know that also these phenomena come in several varieties. Looking back, it may seem like yesterday when we were young, whereas it seems to take forever until next week's film arrives in the cinema (and that, I know from personal experience, is something not only kids complain about). Time appears to fly as long as we enjoy ourselves and it seems to drag when we are understimulated. Obviously, in such cases, it is not chronological time that stretches and squeezes but psychological time .

Time distortion s such as these are based on high-level judgements about duration, which primarily depend on memory, expectation and other psychological factors. That also holds true for Alice's paradoxical experience of spending a day's worth of adventures in Wonderland, which, in the context of the story's 'reality', lasted no longer than an hour. By contrast, the phenomenon of psychological time suddenly speeding up or slowing down, such as experienced by my patient when he walked towards the bus stop, is probably rooted in mechanisms at an intermediate level of the time-perception network. It is controlled neither by the internal master clock nor by DNA expression at the cellular level, but rather by a process at a level in between. It probably involves neuron populations that are also activated when we are about to crash our car into a tailback, or—on a lighter note—are immersed in the action of attempting to score a winning goal at soccer. We all know situations like these, where we are so acutely aware of what is happening, be it in a calamity or in some other high-pressure situation, that we experience everything in astonishing detail, as if it were filmed with a high-speed camera, and played back to us in slow motion. Such changes in the apparent speed of time are called instances of *protracted duration* (for an overview of the various types of time distortion, see Appendix A, Table A.3). Physiologically, protracted duration is intricately linked to the amygdala , an almond-shaped structure in the deeper, evolutionarily older part of the brain that is involved in threat

detection and hence in generating the sensation of fear and preparing the organism for action. Under frightening circumstances, the amygdala is believed to contribute to the formation of memories that are phenomenologically richer, and thus ‘denser’ than ordinary memories, which we therefore tend to recall afterwards as being stretched out over a longer period of time [26]. That is one hypothesis, at least, which begs the question of whether protracted duration is something we experience only in retrospect, or whether we also experience it in the moment itself. I am inclined to think the latter, although more research is needed to cast a proper light on that.

So, within the context of the widely disseminated network for time perception, the amygdala is believed to play a key role in the mediation of protracted duration. Whether that also holds true for the opposite phenomenon (i.e. the quick-motion phenomenon, where everything appears to rush by at an amazing speed) is as yet unknown. However, in addition to the amygdala, numerous other parts of the network for time perception are reported to be of importance.³ Since this area of research has only recently started to gain wider attention, many other parts of the network will probably be added to this list in the future, and different types of time distortion will probably be explained in terms of different states of the network as a whole. Moreover, it is feasible that there may prove to be a structure comparable to visual association cortex, which provides us with a unified sense of time on the basis of all those clocks ticking away inside and outside our body.⁴

5.3 Underlying Disorders

Now that we have looked at the visual, somatosensory and temporal sensory modalities, and have a general idea of the various levels at which they can be compromised when a person has Alice in Wonderland syndrome, it is time to address the question of *how* these lower-level and sometimes higher-level circuits can be erratically switched on and off. How is it possible that cortical columns sometimes selectively stop working? Or all of a sudden show activity when they shouldn’t? And what kind of process is capable of altering our body image in such a way that extra fingers appear to be growing out of our hand? Or of altering the function of the brain’s network for time perception? After all, we do not merely want to

know *which* parts of the perceptual network are responsible for causing the symptoms characteristic of Alice in Wonderland syndrome, we also wish to know *how* they can become so disabled that they stop doing what they should. There are numerous reasons for that to happen, some of which may sound rather ominous, especially if you have first-hand experience with perceptual distortions yourself. However, before we go on to the more serious conditions, let us see what the British neurologist MacDonald Critchley (1900–1997) had so wisely observed, even before Todd had published his paper on Alice in Wonderland syndrome. Having encountered numerous patients with visual distortions, Critchley wrote that,

Metamorphopsia is by no means confined to patients with organic focal disease of the brain. Indeed, most cases occur in quite different circumstances. Some of them are met within normal, though sensitive, aesthetic and introspective individuals [29].

Critchley was an extraordinarily gifted scientist who published more than 200 scientific papers and numerous books. Much of what he wrote was based on his clinical insights rather than on fancy technological innovations. In this case, too, it was his clinical insight that had led him to conclude that the majority of metamorphopsias arise in the absence of some organic focal disease of the brain. Obviously, the brain is always involved in mediating them, but the reassuring message here is that they need not always be caused by serious pathology. That said, whether it is also true that persons experiencing metamorphopsias tend to be sensitive, aesthetic and introspective in nature, as Critchley suggested, is something I would not dare to vouch for. Curiously, the people I have met at my outpatient clinic certainly fit this description; however, contrary to Critchley, I would argue that one needs to be sensitive, aesthetic and introspective to be able to *describe* these phenomena and to seek adequate help for them. My guess would be that individuals with less delicate personalities stand an equal chance of experiencing distortions of whatever kind, but that many of them lack the skills to verbalise what they perceive, and hence fail to point healthcare professionals in the proper direction.

Of course, that does not discredit the first part of Critchley's claim, involving the absence of organic focal brain disease in many instances of metamorphopsia. The exact reason why the brain sometimes produces such

perceptual ‘glitches’ while nothing else appears to be wrong, is something that we do not fully understand, but we do know that Critchley was spot-on with his observation and that it happens in fairly large numbers of people from what we may rightfully call ‘the healthy population’. I mean, how much healthier can one be than a Japanese high school student, a representative of one of the healthiest populations in the world who, moreover, belongs to the healthiest possible age group?

During the 1980s, the Japanese psychiatrists Kazuhiko Abe and Takashi Suzuki from Kitakyushu University performed a survey among 1480 adolescents aged 13–18 years. They had recruited these teens from a number of schools in the vicinity of their university and asked them whether they had ever experienced macropsia or micropsia . To that question, 5.6% of the boys and 6.2% of the girls answered affirmatively [30, 31]. These relatively high numbers baffled the two researchers, who had not expected this and, probably because of that, had not anticipated asking the students under what circumstances they had seen things becoming smaller or larger.

A few years later, Abe headed a second survey and made up for this omission. This time the Japanese researchers studied an even larger group, consisting of 3224 pupils attending high schools (again, 13–18 years of age) whom they asked whether they had experienced macropsia, micropsia or time distortion s during the 6 months preceding the survey. Even though the time frame of this second study was limited to only 6 months, they found a prevalence rate of 3.8% for micropsia, 3.9%for macropsia, 2.5% for protracted duration and 1.3% for the quick-motion phenomenon [32]. Having asked a subgroup of these high school students about the circumstances leading up to their perceptual distortion s, it now became clear that these were relatively common—not to say banal—in nature. Remarkably, none of the students mentioned (or dared to mention) the use of illicit substances . What is more, none of them admitted to glue-sniffing , a cheap method to get high that is known to school kids all over the world. What they did mention, however, was fever (14%), the moment preceding a headache (5%), the moment before falling asleep (11%), the moment of awakening (1%) and fatigue (1%).

Thus, Abe and his group were the first to provide empirical corroboration for Critchley’s clinical observation that metamorphopsias do indeed occur quite frequently in the absence of organic focal brain disease.

However, the most remarkable conclusion that can be drawn from these studies is not the prevalence rates themselves, but the realisation that the actual rates in the general population must be much, much higher. The reason to suspect this is that Abe and his group focussed solely on two types of metamorphopsia and two types of time distortion, whereas numerous other types can be experienced. Moreover, by studying a relatively young group and allowing the second group to mention only symptoms experienced during the past 6 months, the researchers had limited the scope of their survey to such an extent that it did not even come close to what we call ‘exploring the lifetime prevalence rate of symptoms’ (i.e. the number we get when we take a person’s whole life into account). With an average life expectancy of 83.7 years (which is the highest in the world [33]), these Japanese youngsters had plenty of time left *after* the surveys to experience perceptual distortions and to thus increase their lifetime prevalence rates.

That the numbers presented by Abe’s group were indeed extremely prudent was later confirmed by a Finnish study carried out among 297 individuals in the general population with a median age of 25.7 years, who showed prevalence rates of 30.3% for teleopsia (i.e. seeing objects further away than they are), 18.5% for dysmorphopsia (i.e. seeing straight lines as wavy), 15.1% for macropsia and 14.1% for micropsia [34]. Again, the mean age of the population under study was relatively low (although a little higher than that in the Abe studies) as was the number of metamorphopsias taken into account. However, the rates established by this Finnish group were much higher than those by the Abe group, indicating that a study that would take into account *all* known types of perceptual distortion can safely be expected to yield even higher rates—substantially higher, I would say.

What we learn from this, is that metamorphopsias and other perceptual distortions are not rare at all, and that they may be experienced under surprisingly ordinary circumstances such as falling asleep, waking up, being tired or having a fever. Of course, I already knew the latter from my own episode of fever in that bamboo hut in Thailand and from my daughter’s experience with the flu. Apparently, perceptual distortions can also be evoked by changes in one’s body temperature. This I know from an American colleague of mine, who had the exceptional habit of swimming in frigid water during the winter. Of course, he was well aware that one’s body temperature drops quickly under such circumstances, making one shiver

and turning the skin pale and cold. What he also knew is that the cold is no longer felt when the body temperature drops even further, making it seem as though things are getting better while one is insidiously approaching the dangerous stages of hypothermia [35]. That it never got that far, for my colleague, was because he used to get out of the water as soon as he started to see all straight lines as being wavy. As he wrote to me,

When I once swam in Lake Michigan in Chicago in winter, I told the person who was watching me that I knew it was time to get out because all of the skyscrapers looked like they were rubbery and wiggling.

In effect, my heroic colleague employed the onset of dysmorphopsia as a warning sign for him to get out of the water, thus undoubtedly saving himself from freezing on more than one occasion. I have never heard of any other person doing such a thing, but it made me wonder how else perceptual distortions are perhaps put to practical use.

A second lesson to be learned from the population studies is that perceptual distortions are often fleeting and harmless in nature, or else the young people participating in those studies would probably have gone on to seek help, or would at least have been encouraged to do so by their parents. In contrast to Paul, who became so miserable with seeing everything as slanted for such a long time that he considered life no longer worth living, most people do not seem to be strongly affected by their perceptual distortions. And yet we have seen that some of them do, in the sense that they experience recurring or even continuous symptoms, are frightened by them and may be prevented by them from functioning normally. Willem used to send me reports of his experiences through email from time to time, especially when he was on a new type of medication or when the course of his symptoms had taken an unexpected turn. I translated one of those reports, and present it here (with his permission) to illustrate how deep the rabbit hole may go sometimes:

I walked towards the bathroom to brush my teeth there. I felt pretty OK; today had not been my best day, but on the whole I had been able to work. Once inside the bathroom, I looked at the mirror and saw my left eye go down immediately. Normally it takes a while for

that to happen, but now it was almost instantaneous. I cannot tell in a proper chronological order what happened next. It was as though I lost track of time completely. I got into a kind of trance , and my face started to slide. Now and then my eyes, nose, and mouth clung together with my eyebrows, like some sort of pulsating T, which could move freely across the surface of my face. My face often changed colour; sometimes it was completely in the colour of my skin, sometimes blue, sometimes grey, but more often yellow/green, radiating outwards. Even my head became distorted: my moustache and beard flowed in all directions and displayed colourful auras , assuming different positions all the time, as if someone was wildly combing my hair in all directions. I was in a slideshow, looking at myself, but then at alternative versions. It felt as if the versions that I saw, potentially stemmed from a different dimension. It felt a bit like an attack I had had two years ago, when I saw the pattern on my shoes change, and thought that I had ended up in a different timeline. Sometimes my eyes separated themselves from my face, in a way that had happened before. However, this time they sometimes totally disappeared, with a strong focus on [the place] where the eye sockets start, adjacent to the bridge of the nose, where the depth contrast is very high. It was as if I had a very well-defined nose, but no eyes. Sometimes I saw myself completely normal again, albeit with a vague focus on my eyes. I saw my pupils become smaller and larger, independently of each other. There were also moments when it seemed as if my eyes kept looking sideways all the time, like some neurotic lizard. After a while my eyes got completely separated and looked straight back at me, lengthened a bit in a vertical direction, and with a 'religious' gaze, the way you see in Medieval icon paintings. I felt something deep going through me, it felt really important, although absolutely without any form of positivity or negativity. The mirror's edges disappeared; behind me the tiles went on endlessly, and I looked straight at myself, into my soul. I was terrified. After that, the impressive feeling abated a bit, and everything appeared to be less distorted and normal again, until I noticed that every time I blinked, a certain part of me 'flipped'. It felt as if I was being mirrored in a horizontal direction, but not quite. The drawing on my T-shirt remained the same - however, the rest

didn't. It was as if I blinked between two versions of myself, the whole time. This went on for a while, until my face got weirder and more distorted again with each blink. My lips became extremely angular, as though someone had used a ruler to draw its essential shapes, and my nose had been turned completely askew. My skewed nose and mouth tried to escape to the left, but they didn't get far. My face fell apart in a bunch of colour areas, the way a painter would see it. My face was divided perfectly into two colour areas, yellow and red, with a battleground on the bridge of my nose, where red and yellow kept going to and fro all the time to conquer the area. I tried to move my hands; I was a bit more in control by then - and had still not brushed my teeth; but I did not get any further than in front of my head. My hand appeared to be very large, and even though I counted five fingers, there seemed to be too few of them. After that only the distal two phalanges became yellow, the rest red, and they started to stretch themselves to a single height. Normally [the tips of] your fingers form a kind of circular shape, but this time they forced themselves into a straight line, after which they started duplicating. First, extra fingers started to grow, until it was a hand with fifteen fingers; after that, so many that I couldn't even count them anymore; it was as though there were an infinite number of pulsating fingers on my hand. It had practically become a shovel. After that I suddenly had five fingers again, which changed into yellow, pointy teeth, like those of a rat or a guinea pig. Several times my T-shirt (grey, with a drawing on it of a squid in a Mason jar) changed wildly, too; sometimes the squid's tentacles were moving; sometimes, when I looked at the squid's eyes, it seemed as though my own head (almost out of the picture) was full of eyes (so I didn't do that too often); mostly, however, it was a huge, dark-grey storm. There were moments when very long, colourful trails were visible behind contrast points in my face and on my body, especially my beard, my eyes, and the creases in my T-shirt. The T-shirt did move along, but in parts, in 'groups of pixels', the way a video-compression algorithm would do that. I also saw myself breathe, but it seemed as if that only happened in a square of 15×15 centimetres on my belly, as though it had been cut out. A bit later on, my whole body divided itself into tiny, uniformly coloured triangles, like a

kind of mosaic. I stood with my hands resting on the sink, and they changed direction the whole time, even though I did not attempt to do anything with them. In the end I had mustered enough courage to start brushing my teeth, and at last I succeeded, albeit with effort. I closed my eyes, and immediately got confused. During a lightshow of stains and flashes and a washing-machine feeling, I barely succeeded to get the job done, while my toothbrush changed its size inside my mouth, which was extremely confusing. When I opened my eyes to clean the sink, everything was completely green. Slowly, a deep purple blended into my field of vision, which started to mingle with the patterns of scale accumulation on the dimension-stone surface of the sink. The circles became stains, and at some point, it seemed as if a huge amount of paint slithered slowly into the direction of the sink hole. After a few more facial distortions (as described above), I was able to leave the bathroom, although I felt extremely upset. I believe I had been inside for three quarters of an hour in real time, but at some point, I had lost track to such an extent that I thought that it would never stop, especially when things had turned 'religious'. Most of all, I doubted what was real now; the mirror is something my brain finds very confrontational, or something like that. In the future, when I have my own house, I will invest in a mirror that I can turn off

Please feel free to give yourself some room to breathe here, and let the whole story sink in.

This was not some scene from Marvel's *Doctor Strange* .

This was Willem, trying to brush his teeth.

This is what happened to him on a random evening, in the safety of his home, without any LSD , without any other illicit substances and even without a drop of alcohol —just his brain playing tricks with him and making him fall through the cracks of what we call reality. Luckily for him, he had had a pretty good week, and—as he had told me at the time—he had felt so clear-headed throughout the week, that he had been able to do a lot of work at school. Moreover, after a good night's rest, this whole Pandora's box of phantasmagorical experiences had closed itself off again (for the time being) as if nothing had happened to him. But something *had* happened to him of course, and it had frightened him a great deal. We may

all have wished at some point in our lives for a mystical experience to happen that would bring us closer to ourselves, to God , or to whatever metaphysical being or energy we believe in; however, on the basis of this experience alone, Willem would undoubtedly chime in with my prior warning: let us please be careful what we wish for. This rollercoaster ride of his was enough to make his world shake on its foundations and fill him—who had such splendid insight into his situation—with gnawing, existential doubt. What is real? Answer that simple question, while your toothbrush is getting larger and smaller inside your mouth, your face is staring back at you from the mirror with eyes all over it, your hair is blowing back and forth like corn in a hailstorm and the printing on your T-shirt is alive with writhing tentacles and all. Perhaps that is the most frightening aspect of Alice in Wonderland syndrome: that it can make us doubt the most simple, fundamental things about reality, which we had taken for granted since we were little and thought we could be sure of. Intimately connected with that existential fear is another existential fear, namely, that symptoms such as these may indicate some serious disorder underlying it. What if this is a sign that one is psychotic or demented? What if it indicates that there might be a tumour in one's head? It is because of possibilities such as these that we need to be aware of the various brain diseases and other conditions that are sometimes responsible for producing the symptoms of Alice in Wonderland syndrome.

Table A.4 (Appendix A) provides an overview of all the causes of Alice in Wonderland syndrome that we currently know of. It is based on case descriptions and case series of individuals who suffered so severely from their perceptual distortions that they were brought to the attention of clinicians and, in nearly half of the cases, also required treatment. Judging by the publications from which they stem, in children, the most frequent cause of such severe cases of Alice in Wonderland syndrome is encephalitis , or inflammation of the brain. Encephalitis is mostly caused by a virus (Epstein-Barr virus being the most prevalent one in the case descriptions), although it can also be caused by a bacterium or an autoimmune disorder .

In adults, the most prevalent causes of severe Alice in Wonderland syndrome are epilepsy and migraine . As we saw, some authors think that Charles Dodgson suffered from both. Although the clinical presentation of these conditions tends to differ (migraine being exemplified by severe, unilateral headaches , and epilepsy by tonic-clonic seizures), there are

numerous atypical presentations that show considerable overlap. The classic explanatory model for migraine is the spreading-depression theory, which states that a wave of excitatory electrophysiological activity propagates across the cerebral cortex with a speed of several millimetres per minute, followed by a wave of inhibitory activity, which subsequently activates and deactivates adjacent cortical areas, like a forest fire propagating itself through the woods and leaving no signs of life in its wake. A variant of this theory states that it is vascular dilatation and constriction that is responsible for mediating the symptoms characteristic of migraine; however, in both cases the central idea is that of a slow, self-propagating wave through the top layers of the brain. The classical explanatory model of epilepsy is that of electrophysiological activity getting excessively synchronised, thereby affecting an entire cerebral hemisphere in one single surge of electrical impulses—or even both hemispheres, when that ‘surge’ crosses over to the other side. Ever since the pioneering work of the great British neurologist, John Hughlings Jackson (1835–1911)—a contemporary of Charles Dodgson—it has been suspected that also smaller, isolated parts of the brain can be affected by such aberrant surges of electrical activity. Thus, even though the symptoms of Alice in Wonderland syndrome may be experienced in the context of full-blown migraine or epilepsy (as an aura or ‘warning signal’, as an accompanying symptom of the attack itself, or in the aftermath of an attack), they can also be experienced in isolation, without the accompaniment of any headache or tonic-clonic seizure. In such subtle cases, the underlying electrophysiological disturbance is probably limited to a relatively small part of the perceptual network, from where it does not go on to spread to other parts. Thus, not only large brain areas can be affected, as suggested by the classical theories of epilepsy and migraine, but also neuron populations on a much smaller scale, which may moreover be located in deeper regions of the brain, which lie out of the reach of conventional EEG tracings.

Continuing our inventory, the conditions capable of causing Alice in Wonderland syndrome can be divided into eight main groups (Table A.4, Appendix A). Among the truly serious ones are obviously brain tumours and strokes, which we definitely don’t want to have [36–39]. Mostly, however, such alarming cases will present with additional symptoms such as aphasia, paresis or cognitive dysfunction, although it is always wise to remember that, in rare instances, perceptual distortions *may* be the sole

presenting symptom of such severe conditions. Thus, together with neurologist Bas ter Meulen and his colleagues from Amsterdam, I published the dramatic case of a 68-year-old man who started to suffer from isolated metamorphopsias, and subsequently died within 2 months due to Creutzfeldt-Jakob disease, a rare brain disorder for which no treatments are available [40].

Regarding the other possible causes of Alice in Wonderland syndrome, especially worth mentioning is the large group of drug-induced cases, known as *hallucinogen-induced persistent perception disorder* or *hallucinogen persisting perception disorder* (HPPD). Both terms appear to suggest that the diagnosis only applies to cases related to the use of hallucinogens, such as LSD, but in clinical practice it is used for all cases where illicit substances play a role, including cannabis, amphetamines and cocaine. Incidentally, HPPD should not be confused with the acute effects of intoxication. We all know that LSD may cause numerous perceptual distortions, ranging from oversaturated colours, objects that appear to be bent out of shape and walls that seem to breathe to hallucinated tunnels, spirals, honeycombs, lattices, trailing phenomena, somesthetic distortions, time distortions and much, much more; however, HPPD is the condition where such acute effects have worn off, and the distortions nonetheless linger on, or else have returned at a later stage. This may happen even after several years of abstinence. Not everyone experiencing perceptual symptoms after such a long time will make the connection with prior substance use, even though the symptoms themselves are often reminiscent of those experienced during the intoxication phase.

We already met Mr. Salvatore, whose hands appeared to shrink to the size of tiny stumps whenever he put them in his pockets. You will remember that he had been suffering from this type of (partial) microsomatognosia, as well as from derealisation, depersonalisation and various types of metamorphopsia, for over 35 years. What I had not explained before is that his impressive collection of symptoms had begun at the age of 14 while he was smoking cannabis. Even in the Netherlands, with its liberal drugs policy, 14 years is an unusually young age to start with cannabis. But Mr. Salvatore had done so, and had soon experienced what he called '*three horror trips*'. During one of those trips, he had had the overwhelming sensation that his head was shrinking to tiny proportions, while simultaneously his tongue grew enormously in size. It is not hard to

imagine how he must have freaked out at that moment, and how badly he must have wanted the sensation to go away. To his relief, it disappeared as soon as the acute effects of the intoxication wore off, but not long thereafter, he started to get flashbacks of that awkward moment, aggravated by instances of derealisation and depersonalisation. Over the years, the flashbacks of the head-shrinking experience faded away, but they were gradually replaced by somewhat similar experiences involving his hands. In addition, he started to see stationary objects as if they were moving away from him (porropsia), and to experience other metamorphopsias as well. Despite numerous consultations with specialists, and numerous pharmacological and psychotherapeutic treatments, after 35 years, he had only managed to obtain a reduction in symptoms of around 50%.⁵

We also met Mr. Müller, with the recurring sensation of having a fluttering ‘gill’ on his shoulder, who had been unable to work for the past 7 years. This bizarre somatosensory distortion, too, had started out during an episode of cannabis consumption. Having had a stressful time at school, with highly demanding pupils, and difficulty coping with his back pain, he had resorted to smoking cannabis for the first time in his life. Because it had helped him to get the sleep he needed so badly, and had also relieved the pain in his back, it had soon become a daily habit for him. When after 3 months the fluttering sensation had started, he had quit immediately. However, even though he had been completely abstinent ever since and had tried numerous pharmacological treatments, the sensation kept plaguing him several times a day.

Fortunately, HPPD is not always as persistent and incapacitating as it was in Mr. Salvatore’s and Mr. Müller’s case. The characteristic flashbacks and other perceptual symptoms may sometimes resolve spontaneously—after a month, or even after a year [41]. Nevertheless, the burden to those affected is usually substantial, and more often than not, the condition lasts for many years. On the whole, HPPD therefore has the reputation of a chronic, distressing condition that is disappointingly resistant to treatment. Even though the substances that may trigger it are known to act through numerous different neurotransmitter systems, it has been suggested that the serotonergic system may well act as a final common pathway for HPPD [42]. Future studies will have to establish whether that is true and also whether HPPD deserves its reputation of chronicity, although people like Mr. Salvatore and Mr. Müller will be the last to debate that.

A totally different group of causes of Alice in Wonderland syndrome includes those that affect the peripheral rather than the central nervous system. Eye disease is an example of this group of disorders. I was once consulted by a young man who complained that he saw all straight lines as wavy. Not having an Amsler grid ⁶ at my disposal, I took him to the kitchen of our department, where there are tiles on the floor like those in a swimming pool, which could do the trick just as easily. He told me that the floor actually looked like the bottom of a pool because no matter with which eye he looked at them, the grout joints looked startlingly wavy. As he had been suffering from this without a pause for several weeks, I gave him an urgent referral to an ophthalmologist. Dysmorphopsia, as this particular type of metamorphopsia is called, can be caused by retinal ablation. Other causes include myopic maculopathy, age-related macular degeneration, diabetic retinopathy and numerous other eye diseases that have nothing to do with cortical columns and other brain mechanisms involved in the ‘central’ types of metamorphopsia [43]. Likewise, some cases of plagiopsia and kinetopsia can be caused by diseases of the organ of balance in the inner ear.

Sometimes, symptoms of Alice in Wonderland syndrome are also attributed to psychiatric disorders. Thus, there are several publications describing them in the context of mood disorders, dissociative disorders and psychotic disorders [34, 39, 44–46]. However, even though the latter category is my own specialty, I hardly ever encounter people with perceptual distortions in my clinical population of psychotic patients. That said, I may well systematically overlook them in this group because symptoms as subtle as these are difficult to assess when people are too confused, and/or lack the verbal skills to describe them—or else be too consumed by their voices or delusions to pay attention to misperceptions of a subtler type.

Lastly, it is worth mentioning that various psychodynamic interpretations of Alice in Wonderland have been put forward, characterising its symptoms as ‘*psychogenic*’, ‘*transcortical*’ or ‘*hysterical*’ in nature [47–51]. Although there are subtle differences in meaning between these terms, what they convey is that metamorphopsias and other distortions have a mental rather than an organic basis, in the sense that they are mediated by the mind and not the brain. These interpretations take us back to the dualistic models I mentioned before, which state that perception

is a mental event that cannot be equated with brain activity. Since two and a half millennia of philosophical thinking have failed to yield a satisfying solution to the mind–body problem, I don't think it is up to me to provide one. However, if any of these psychodynamic interpretations implies that the mind is capable of producing symptoms of Alice in Wonderland without the involvement of the brain, my background in neurology prevents me from envisioning how that would even be possible. If, on the other hand, they might imply that psychological factors are capable of influencing the brain in such a way that they *promote* these symptoms, I will be the first to agree. As we saw, some people report perceptual distortions when they are fatigued. In addition, numerous other psychological stressors are capable of aggravating such symptoms, and I can imagine that they may even cause them, in the sense that they may set in motion the neural machinery responsible for mediating them. In general, psychological factors have to be reckoned with, even in the context of thoroughly biological disorders. Moreover, some authors found that symptoms characteristic of Alice in Wonderland syndrome can be evoked by meditation [52]. Another case in point is the claim made by Kinnier Wilson (1878–1937), an American-born neurologist who used to work in the UK, who wrote that he himself had experienced episodes of micropsia as a child and that he could terminate them at will [47]. However, even that remarkable claim does not prevent me from thinking that, in his case, too, the symptom itself must have had an organic aetiology. Yet another argument against the alleged possibility of a psychological origin of Alice in Wonderland is that the papers on this topic invariably fail to show that proper adjuvant investigations had been carried out to rule out any underlying organic pathology.

Therefore, in my opinion, it is best to exert proper caution while dealing with psychodynamic interpretations of Alice in Wonderland syndrome and keep in mind that there are numerous smaller and larger circuits within the perceptual network that can cause any of its numerous symptoms, depending on their function within the network as a whole, and triggered by any of the numerous conditions we just discussed.

A century ago, the German psychiatrist Emil Kraepelin (1856–1926) compared the brain to an organ [53]. Of course, the brain *is* an organ, but in this case, Kraepelin meant the other kind, the keyboard instrument. We all know that organs can produce numerous notes, although their number is limited by the number of keys. We also know that the same notes can be

brought forth by different agents, for example, by pressing the keys with one's fingers, as one usually does—but also with one's toes, or even with a hammer or a bath duck, if one would like to. In other words, whatever we do, what we can expect from organs is a limited number of predetermined sounds. After all I have said about not comparing the brain to a video camera or even to a computer, this may strike you as a somewhat simplistic metaphor. And yet I like it, since it sums up in a nutshell how the brain can be affected by numerous pathological agents, which are nonetheless capable of calling forth no more than a finite number of stereotypical reactions, depending on the physiological function of the part of the brain affected. This certainly holds true for Alice in Wonderland syndrome, with its numerous underlying causes—including, of course, psychological factors .

References

1. Wachowski L, Wachowski L (1999) *The matrix*. Warner Bros, Burbank
2. Lanska JR, Lanska DJ (2013) Alice in Wonderland syndrome: somesthetic vs visual perceptual disturbance. *Neurology* 80:1262–1264
[\[PubMed\]](#)
3. Hubel DH, Wiesel TN (2005) *Brain and visual perception*. Oxford University Press, Oxford
4. Hubel DH, Wiesel TN (1959) Receptive fields of single neurones in the cat's striate cortex. *J Physiol* 148:574–591
[\[PubMed\]](#)[\[PubMedCentral\]](#)
5. Horton JC, Adams DL (2005) The cortical column: a structure without a function. *Philos Trans R Soc Lond B Biol Sci* 360:837–862
[\[PubMed\]](#)[\[PubMedCentral\]](#)
6. Kamitani Y, Tong F (2005) Decoding the visual and subjective contents of the human brain. *Nat Neurosci* 8:679–685
[\[PubMed\]](#)[\[PubMedCentral\]](#)
7. Alink A, Walther A, Krugliak A, Kriegeskorte N (2017) Local opposite orientation preferences in V1: fMRI sensitivity to fine-grained pattern information. *Sci Rep* 7:7128
[\[PubMed\]](#)[\[PubMedCentral\]](#)
8. Hubel DH, Wiesel TN (1970) Cells sensitive to binocular depth in area 18 of the Macaque monkey cortex. *Nature* 227:41–42
9. Ierodiakonou K (2005) Plato's theory of colours in the *Timaeus*. *Rhizai J Anc Philos Sci* 2:219–233
- 10.

Prior RCA (1878) Was Homer colour-blind? *Nature* 19:119–120

11. Locke J (1690) *An essay concerning human understanding*. Thomas Bassett, London
12. Nietzsche F (1878) *Menschliches, allzumenschliches. Ein Buch für freie Geister*. Verlag von E.W. Fritzsche, Leipzig
13. Zeki S (1990) A century of cerebral achromatopsia. *Brain* 113:1721–1777
[\[PubMed\]](#)
14. Zeki S (1991) Cerebral akinetopsia (visual motion blindness). A review. *Brain* 114:811–824
[\[PubMed\]](#)
15. Pözl O (1928) *Die optisch-agnostische Störungen*. F. Deuticke, Leipzig
16. Bay E (1953) Disturbances of visual perception and their examination. *Brain* 76:515–550
[\[PubMed\]](#)
17. Willanger R, Klee A (1966) Metamorphopsia and other visual disturbances with latency occurring in patients with diffuse cerebral lesions. *Acta Neurol Scand* 42:1–18
18. Enoch JM (2006) History of mirrors dating back 8000 years. *Optom Vis Sci* 83:775–781
[\[PubMed\]](#)
19. Ramachandran VS, Blakeslee S (1998) *Phantoms in the brain. Probing the mysteries of the human mind*. William Morrow, New York
20. McGonigle DJ, Hänninen R, Salenius S, Hari R, Frackowiak RSJ, Frith CD (2002) Whose arm is it anyway? An fMRI case study of supernumerary phantom limb. *Brain* 125:1265–1274
[\[PubMed\]](#)
21. Blom JD (2014) When doctors cry wolf. A systematic review of the literature on clinical lycanthropy. *Hist Psychiatry* 25:87–102
[\[PubMed\]](#)
22. Blom JD, Neven A, Aouaj Y, Jonker B, Hoek HW (2010) De coenesthesiopathieën. *Tijdschr Psychiatr* 52:695–704
[\[PubMed\]](#)
23. Hamed SA (2010) A migraine variant with abdominal colic and Alice in Wonderland syndrome: a case report and review. *BMC Neurol* 10:2
[\[PubMed\]](#)[\[PubMedCentral\]](#)
24. Eagleman DM (2008) Human time perception and its illusions. *Curr Opin Neurobiol* 18:131–136
[\[PubMed\]](#)[\[PubMedCentral\]](#)
25. Üstün S, Kale EH, Çiçek M (2017) Neural networks for time perception and working memory. *Front Hum Neurosci* 11:83
[\[PubMed\]](#)[\[PubMedCentral\]](#)
26. Hickie IB, Naismith SL, Robillard R, Scott EM, Hermens DF (2013) Manipulating the sleep-

wake cycle and circadian rhythms to improve clinical management of major depression. *BMC Med* 11:79
[PubMed][PubMedCentral]

27. Zhang J, Wang G, Jiang Y, Dong W, Tian Y, Wang K (2012) The study of time perception in migraineurs. *Headache* 52:1483–1498
[PubMed]
28. Craig AD (2009) How do you feel--now? *Nat Rev Neurosci* 10:59–70
[PubMed][PubMedCentral]
29. Critchley M (1953) *The parietal lobes*. Edward Arnold, London
30. Abe K, Suzuki T (1986) Prevalence of some symptoms in adolescence and maturity: social phobias, anxiety symptoms, episodic illusions and idea of reference. *Psychopathology* 19:200–205
[PubMed]
31. Abe K, Suzuki T (1986) Age trends of social phobias, anxiety symptoms, morning dysphoria, early awakening and episodic illusions in 9-60 years of age. In: Shagass C (ed) *Biological psychiatry (development in psychiatry)*. Elsevier, New York, pp 607–609
32. Abe K, Oda N, Araki R, Igata M (1989) Macropsia, micropsia, and episodic illusions in Japanese adolescents. *J Am Acad Child Adolesc Psychiatry* 28:493–496
[PubMed]
33. World Health Organisation (2017) *World health statistics 2017: monitoring health for the SDGs*. World Health Organisation, Geneva
34. Lipsanen T, Lauerma H, Peltola P, Kallio S (1999) Visual distortions and dissociation. *J Nerv Ment Dis* 187:109–112
[PubMed]
35. Zonnenberg C, Bueno de Mesquita JM, Ramlal D, Blom JD (2017) Hypothermia due to antipsychotic medication: a systematic review. *Front Psych* 8:165
36. Bender MB, Kanzer MG (1941) Metamorphopsia and other psychovisual disturbances in a patient with tumour of the brain. *Arch Neurol Psychiatr* 45:481–485
37. Geyer K-H (1963) Zentrale Störungen des Formensehens. Zur Pathogenese der Metamorphopsie. *Dtsch Z Nervenheilkd* 184:378–387
[PubMed]
38. Ceriani F, Gentileschi V, Muggia S, Spinnler H (1998) Seeing objects smaller than they are: micropsia following right temporo-parietal infarction. *Cortex* 34:131–138
[PubMed]
39. Camacho Velasquez JL, Rivero Sanz E, Tejero Juste C, Suller Marti A (2016) Síndrome de Alicia en el país de las maravillas en patología cerebrovascular. *Neurologia* 31:418–420
[PubMed]
40. Naarden T, ter Meulen BC, van der Weele SI, Blom JD (2019) Alice in Wonderland syndrome as

a presenting manifestation of Creutzfeldt-Jakob disease. *Front Neurol* 10:473
[PubMed][PubMedCentral]

41. Abraham HD (1983) Visual phenomenology of the LSD flashback. *Arch Gen Psychiatry* 40:884–889
[PubMed]
42. Litjens RP, Brunt TM, Alderliefste GJ, Westerink RH (2014) Hallucinogen persisting perception disorder and the serotonergic system: a comprehensive review including new MDMA-related clinical cases. *Eur Neuropsychopharmacol* 24:1309–1323
[PubMed]
43. Midena E, Vujosevic S (2016) Metamorphopsia: an overlooked visual symptom. *Ophthalmic Res* 55:26–36
44. Bui E, Chatagner A, Schmitt L (2010) Alice in Wonderland syndrome in major depressive disorder. *J Neuropsychiatr Clin Neurosci* 22:352
45. Coleman SM (1933) Misidentification and non-recognition. *J Ment Sci* 79:42–51
46. Blom JD, Looijestijn J, Goekoop R, Diederens KMJ, Rijkaart A-M, Slotema CW, Sommer IEC (2011) Treatment of Alice in Wonderland syndrome and verbal auditory hallucinations using repetitive transcranial magnetic stimulation. A case report with fMRI findings. *Psychopathology* 44:337–344
47. Wilson SAK (1916) Dysmetropsia and its pathogenesis. *Trans Ophthalmol Soc UK* 36:412–444
48. Schneck JM (1961) Micropsia. *Am J Psychiatr* 118:232–234
[PubMed]
49. Schneck JM (1984) Psychogenic micropsia in fact and fiction. *J Am Med Assoc* 251:2350
50. Meyers WA (1977) Micropsia and testicular retractions. *Psychoanal Q* 46:580–604
51. Wiese J (1979) Derealisationsphänomene: Psychophysiologische Untersuchung und psychodynamische Interpretation bei einem 9-jährigen Jungen mit Mikropsien. *Prax Kinderpsychol Kinderpsychiatr* 28:133–136
[PubMed]
52. Civardi C (2015) Meditating for Alice in Wonderland syndrome. Meditation can evoke the same experience as hallucinative child fevers. United Academics. <http://www.united-academics.org/mind-brain/meditating-for-the-alice-in-wonderland-syndrome/>. Accessed 24 Apr 2016
53. Kraepelin E (1920) Die Erscheinungsformen des Irreseins. *Z Gesamte Neurol Psychiatr* 62:1–29

Footnotes

1 To be totally accurate, I must point out that V4 is a bilateral structure and that, therefore, both parts would need to be switched off in order to make us see the world in black and white. When only one is switched off, we see half of the visual field in black and white and the other half in colour.

2 This example of akinetopsia confirms that the brain is not a video camera. For a video camera to create the illusion of smooth movement, it only needs to play back the recorded series of images at the speed of 24 frames per second; for many purposes, even 16 frames per second would be sufficient. The brain, however, needs to switch on V5 to perceive this as smooth movement. No matter how many frames per second we would add, without V5 functioning properly, the net result will be a succession of images rather than a seamless flow.

3 These parts include the basal ganglia, the cerebellum, parietal cortex, extrastriate visual cortex , anterior cingulate cortex, posterior cingulate cortex, premotor cortex and frontal cortex. Of note, the cerebellum has been implicated in timing durations in the milliseconds range rather than the seconds-to-minutes range and found to be compromised in patients diagnosed with migraine, making them significantly overestimate the duration of such short intervals of time [27].

4 A candidate structure for such a ‘receptor function’ in time perception has been suggested by Craig, who described the role of the insula , or the brain’s ‘fifth lobe’, in collecting and integrating information from within the body as a whole, including information that has bearing on the subjective experience of time [28].

5 In a way, Dimitri’s story was similar to that of Mr. Salvatore’s. Even though he had been diagnosed with schizophrenia (whereas Mr. Salvatore had not), he suffered from several types of perceptual aberration that were at least reminiscent of metamorphopsia s. Looking back, they might well have been caused by the illicit substances that he had been using in the past. In Dimitri’s case, they included cannabis (which he had been smoking since the age of 15, in quantities of up to 4 joints a day), XTC , herbal XTC, mushrooms , Ephedra and Salvia . Although he had used the latter substances only occasionally, all in all, it was enough to justify the hypothesis that his metamorphopsias had something to do with his prior substance abuse . He, too, had been treated for years on end with various medicines. This had helped him to keep his paranoid delusions and disorganisation under control but, as we saw, not the distortions .

6 The Amsler grid was developed around 1945 by the Swiss ophthalmologist, Marc Amsler (1891–1968). It consists of a card with intersecting vertical and horizontal lines, which is used by ophthalmologists and optometrists to test visual disturbances caused by retinal and macular disorder s.

6. Diagnosis and Treatment

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

How should Alice in Wonderland syndrome be diagnosed? And how should it be treated? For numerous other medical conditions, diagnostic criteria are available from scientific organisations such as the *World Health Organisation*, the *American Psychiatric Association*, the *International League Against Epilepsy* and so on. In addition, these organisations may issue evidence-based guidelines which stipulate how disorders should preferably be treated. However, for Alice in Wonderland syndrome, no such diagnostic criteria and therapeutic algorithms are available. As you probably have guessed, that is because the syndrome has until now hardly been investigated. As a consequence, Alice in Wonderland syndrome is perhaps *the* poster child for orphan disease s: a condition ‘forgotten’ by the powers that be and, therefore, playing no role whatsoever in the canon of medicine.

It is still a mystery what happened to Todd’s own involvement with Alice in Wonderland syndrome once his paper had been published. Did he go on to research his newly designed syndrome? Did he start lobbying or campaigning to give it wider recognition? Did he attempt to define diagnostic criteria or therapeutic algorithms himself? At High Royds Hospital, where he had accepted a position as a consultant psychiatrist by the time his paper appeared in print, he must have gone on to diagnose the syndrome in his patients whenever he saw fit, even in the absence of formal diagnostic criteria. Based on the little that we know about him, I do not

believe that Todd was the kind of man who would have let himself be held back by ‘trivialities’ such as a lack of criteria issued by some official committee. However, after his publication of the Alice-in-Wonderland paper, there is virtually nothing more to be found on the topic.

However, there is one additional publication, written by an anonymous author, which appeared a full year after Todd’s own paper, in the special Christmas edition of *What’s New*, an in-house company magazine of Abbott Laboratories, a US-based pharmaceutical multinational. In it, Alice in Wonderland syndrome was hailed as a new term, which had been proposed ‘not only because it is pertinently descriptive but also because it points to the fact that Lewis Carroll suffered from migraine’ [1]. For the rest, the text reiterated the contents of Todd’s 1955 paper [2] and offered some quotes which suggest that the godfather of Alice in Wonderland syndrome may have been interviewed in person for the occasion, along with Lippman (the neurologist who had suggested that Dodgson had suffered from migraine [3]), although the two of them said nothing there that they had not already stated in their respective papers.

If this was Todd’s attempt to get media exposure for his newly coined syndrome, it may not have been the most felicitous choice. After all, we may ask ourselves, who, outside the circle of Abbott Laboratories’ employees and clients, would have read the article at the time, and how seriously they would have taken a ‘newly discovered disease’ presented in a festive issue of their company’s glossy. Whatever reasons Todd may have had for agreeing to the piece—which came adorned with four reproductions of Tenniel’s illustrations, as well as a moody gouache of a man in classic dress, visibly weighed down by sorrow—it can hardly have been some grand scheme aimed at launching Alice in Wonderland syndrome into mainstream medical thinking, or a bid to convince scientific committees to include it in their classifications.

Be that as it may, Todd himself was apparently more interested in exploration than in implementation and may, therefore, not have been unduly bothered by the fact that this was not a ‘serious’ journal. His writings indicate that his insatiable appetite for new exotic syndromes had soon led him into different directions. As a consequence, in his later works, he only occasionally described patients with perceptual distortions. Moreover, on the few occasions that he did describe them, it was invariably in the context of some other disorder that had kindled his interest. In one of

his papers from the 1960s, for instance, he relates how a female patient had perceived goods in a shop window as being distorted [4]. As Todd tells us, this was due to an insulin-producing tumour, a new, exciting condition that he was into at the time. And that was what the paper was about. As far as we know, he never doubled back on his original publication to expand or refine his concept of Alice in Wonderland syndrome or to formulate diagnostic criteria for it. Because no one else has done so since, we might as well have a go at it ourselves: that is, to find out what such criteria should preferably look like, and to see what can be said about treatment options—provided that treatment is necessary in the first place.

6.1 Diagnosis

The role of Alice in Wonderland syndrome in current diagnostic classifications is negligible. In my field, the most influential ones are the *International Classification of Diseases and Related Health Problems* (ICD-10 [5])—which covers medical conditions in general, including neurological and psychiatric disorders—and the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5 [6]), which covers only psychiatric disorders. Neither of the two systems features Alice in Wonderland as a diagnostic category. The ICD-10 mentions several types of metamorphopsia under the heading of *Other Visual Disturbances*, including ‘visual distortion’, ‘polyopia’, and ‘blurred vision’. What the DSM-5 does feature is a diagnostic category called *Hallucinogen Persisting Perception Disorder*, which I mentioned before as the late-onset, drug-induced variant of Alice in Wonderland syndrome. Elsewhere, the DSM-5 also mentions the symptoms of ‘two-dimensionality or flatness, exaggerated three-dimensionality, or altered size or distance of objects (i.e. macropsia or micropsia)’, but only in the context of a condition called *Depersonalisation/Derealisation Disorder*. In sum, these two major classifications are of little help when we wish to assess the presence or absence of Alice in Wonderland syndrome. This is not surprising, given the fact that the scientific committees issuing them need data from empirical studies to work with, which for this topic have only recently begun to accumulate.

So, what should the diagnostic criteria for Alice in Wonderland syndrome look like? First and foremost, what is needed is a set of criteria that allows clinicians to distinguish cases from non-cases. Since the

hallmark symptom of Alice in Wonderland syndrome is perceptual distortion, the visual and other distortions listed in Tables A.2 and A.3 (Appendix A) would qualify perfectly well as primary diagnostic criteria.

Second, what is needed is a criterion by which to establish whether such cases are also clinically relevant. For the purpose of diagnosing major depressive disorder, for example, the DSM-5 stipulates that five out of nine possible symptoms of depression need to be present. Likewise, we need to decide how many types of distortion need to be present to justify a diagnosis of Alice in Wonderland syndrome. Since even a single symptom may be clinically relevant, as we saw in Paul's case of plagiopsia (seeing all things as slanted), in Mrs. van Nuys' case of prosopometamorphopsia (seeing faces change into dragon faces), and in Mr. Müller's case of somesthetic distortion (i.e. the 'fluttering gill' on his shoulder), I would argue that it is not the number of symptoms but rather their impact on people's lives that should qualify as such a criterion for clinical relevance.

Third, clinicians need to be able to score adjuvant symptoms of Alice in Wonderland syndrome, such as derealisation and depersonalisation—which are not necessarily part of the syndrome itself, since they are also experienced in the context of dissociative identity disorder and other conditions. To that end, we might add a *specifier*, that is, a clause that covers adjuvant symptoms which do not belong to the disorder's core symptoms but are, nevertheless, considered to be of clinical importance.

In the fourth place, clinicians need to be able to specify whether distortions are unimodal or multimodal in nature, i.e. whether they are experienced in one or more sensory modalities, since that may say something about their origin and may also have repercussions for treatment.

In the fifth place, it may be useful to have a specifier for the course of symptoms over time, since this may say something about the underlying disorder (i.e. paroxysmal or not) and about prognosis (although we already saw that even long-lasting symptoms are sometimes very amenable to treatment). For that purpose, we should have a system that allows clinicians to operationalise for how long the symptoms have been present, whether they have been present continuously or intermittently, and whether or not they are currently in remission.

Sixth—and finally—clinicians will need a specifier that reflects the demonstrable presence or absence of an underlying medical condition, such as encephalitis, migraine, epilepsy, substance abuse and so on, in

accordance with the known possibilities as listed in Table A.4 (Appendix A). This includes so-called ‘peripheral’ causes such as eye disease and labyrinthine disease —plus a category ‘not otherwise specified’ for cases where the aetiology cannot be established with sufficient confidence.

On the basis of these considerations, Appendix C provides a proposal for the full diagnostic criteria for Alice in Wonderland syndrome, to be used in clinical practice and to be taken into consideration for inclusion in international diagnostic classifications.¹

Obviously, the diagnostic process itself stands and falls with proper history-taking. To assess Alice in Wonderland syndrome in clinical practice, clinicians need to be familiar with the numerous types of distortion that can be experienced and also be open to encountering new ones that have not yet been described in the scientific literature. That includes distortions in sensory modalities other than those described by Todd.² While assessing the presence or absence of these symptoms, it may be helpful to use material that enables one to visualise each separate type of distortion.

If one or more symptoms are found to be present, a general psychiatric and neurological examination is mandatory. There may also be a need to carry out auxiliary investigations, such as blood tests, an EEG, an MRI head and, sometimes, also a lumbar puncture, an ophthalmologic examination, and/or an ear-nose-throat examination. It is left to the clinician’s professional judgement as to whether all these are necessary but —when in doubt—my advice would be to carry out those tests. On the basis of the outcome, clinicians will then be able to diagnose cases as Alice in Wonderland syndrome as either:

1. Alice in Wonderland syndrome, central type, in the context of another medical condition (to be specified if known);
2. Alice in Wonderland syndrome, peripheral type, in the context of another medical condition (to be specified if known) or
3. Alice in Wonderland syndrome not otherwise specified (NOS)

6.2 Treatment

Before coming up with a treatment plan, clinicians always need to assess the need to treat. This is a general rule in medicine but one that cannot be overemphasised, especially in the context of Alice in Wonderland syndrome. After all, the benefits of treatment do not always outweigh the risks or side effects, and sometimes people may have other reasons—mostly of a personal or a religious nature—to refrain from treatment, or simply wish to postpone it for a while. Since not all symptoms of Alice in Wonderland are caused by some life-threatening or incapacitating medical condition, and not all of them are extremely burdensome, reassurance and explanation may sometimes suffice. Moreover, in cases without a demonstrable cause, it may be worthwhile to wait for some time and observe the natural course of the symptoms (as Paul's mother had so wisely chosen to do for her 6-year-old son).

When treatment is considered necessary and/or desirable, it is unfortunate that we have no evidence-based protocols to rely on. However, on the basis of the literature—and my own clinical experience—I would recommend to aim treatment at the underlying cause, if known and if possible, or else at the most likely cause. When symptoms come and go in a paroxysmal fashion, for example, the way they do in the context of epilepsy, it may be worthwhile to start with antiepileptics, even in the absence of positive findings on an EEG. The clinician's expertise should be leading in such cases. Thus, symptoms suspect of epilepsy should be treated with antiepileptics, those suspect of migraine with migraine medications (although the perceptual symptoms of migraine are often amenable to antiepileptics, too), those suspect of infectious disease with antibiotics or antiviral medications and so on. Symptoms caused by a stroke or a brain tumour obviously warrant specialised neurological, neurosurgical or—in the latter case—oncological treatment. In cases of intoxication, abstinence is the first step to be taken, although that may not always suffice, and symptomatic treatment may also be necessary. Of all the clinically relevant cases described in the literature, some 50% were treated, whereas the other 50% were left untreated, which, in many cases, resulted in spontaneous remission. Incidentally, that does not mean that we might as well refrain from treatment altogether, since the treatment group comprised patients whose underlying disorders tended to be more severe, as well as patients who were more severely burdened by their perceptual distortions.

For the purpose of symptomatic treatment, several medications can be prescribed. In the literature, success has been reported mainly with antiepileptics, cholinesterase inhibitors, and benzodiazepines. Since benzodiazepines tend to be highly addictive, my advice would be to refrain from prescribing them, unless one wishes to use them to assess their effects on the threshold for epileptic activity. One of my patients, for example, found that he could temporarily alleviate his metamorphopsias by using the benzodiazepine, oxazepam. When I prescribed an antiepileptic instead, and tapered off the oxazepam, he became symptom-free and no longer ran the risk of oxazepam addiction. Antidepressants and antipsychotics may be prescribed when the underlying disorder is depressive disorder or psychosis, respectively; however, in the absence of such disorders, these medicines seem to be of little value. As we saw, Dimitri's psychotic symptoms had gone in remission with the aid of the antipsychotic, clozapine, whereas his metamorphopsias had not. Another reason to be cautious with antipsychotics in the context of Alice in Wonderland syndrome is that these medicines may lower the threshold for epileptic activity and may, thus, aggravate perceptual distortions [7].

Like I said, before initiating treatment, it is paramount to assess the patient's need to be treated. In addition, one should point out that no treatment protocols exist for Alice in Wonderland syndrome, and that the medicines to be prescribed for *symptomatic* treatment are therefore off-label. Finally, clinicians would be wise to manage their patients' expectations in advance, since outcome studies are practically non-existent, and it is hard to predict therapeutic success in advance.

In my own experience, the results of the above-mentioned treatments for Alice in Wonderland syndrome vary widely. At first, Dimitri seemed to benefit from the antiepileptic, valproic acid, which I had prescribed, but after we got to know each other a bit better, it transpired that his metamorphopsias had hardly diminished—if at all. Yet he declined my offer to try any other type of medication, since the symptoms no longer really bothered him. As he told me: the way we had discussed the origins of his symptoms as stemming from his brain had not fully convinced him that that was actually the case. Nonetheless, it had diminished his anxiety and made it easier for him not to be distracted by his symptoms.

Mr. Salvatore, whose symptoms had begun during his 'horror trips' while on cannabis, had been searching for more than 30 years for a proper

treatment. What we finally settled on was another antiepileptic, called lamotrigine , which diminished his symptoms by about 50%; however, even after endless fine-tuning, this medicine did not render him symptom-free. Because he feared that this progress might be wasted if he tried any other type of medication he, too, declined my offer to switch to something else.

Mrs. van Nuys, the woman who saw dragon faces, was initially very happy with the valproic acid I had prescribed—but had had to taper it off because of recurring ‘bangs’ during the night. This rare condition, called exploding head syndrome , also plagued her after she had switched to another antiepileptic , gabapentin. Eventually, it was the cholinesterase inhibitor , rivastigmine , which finally stabilised her. Even though she was the patient in my practice with the longest duration of Alice in Wonderland syndrome (having suffered from it for more than 50 years), this treatment was very successful. Seven years after we had first met, she was still very happy with rivastigmine. She went on to experience occasional loud noises during the night, but they bothered her far less than the dragon faces, which had practically disappeared.

The same held true for Ms. Rembrandt, who had been suffering from almost daily recurring symptoms since early childhood onwards. In her case, the valproic acid I had prescribed had had almost immediate success and, after tweaking the dosage a bit, she had become symptom-free for the first time in over four decades. As she wrote to me a year later: she was still symptom-free by then—and still had to get used to this new ‘ordinary’ life of hers, wondering how other people managed to deal with that after such a long time.

Willem, on the other hand, had no benefit from the valproic acid but subsequently attained a considerable reduction of his unsurpassed collection of perceptual distortions on gabapentin . He was able to resume his education and functioned much better. Because his almost daily attacks had disappeared on the gabapentin but for some mysterious reason had been replaced by weekly occurring attacks that were much more violent in nature, he agreed to change the gabapentin for carbamazepine , yet another antiepileptic. After an initial increase in the number of ‘grand’ attacks (one of which was described by him in the previous chapter), the carbamazepine helped greatly to reduce the number and severity of his attacks.

And Ms. Artemis, the young, bright woman with whom this book began? The one with the squirrel, who saw her desk accessories hovering

silently into the air? What happened to her? Sometimes we cross paths with people who have extraordinary life stories to tell, and sometimes they part from us just as easily. Ms. Artemis was such a person. She had been referred to me by her neurologist, who had examined her and had done all the work-up necessary in such cases. After I had seen her, too, and had explained to her what I thought had been the cause of her symptoms, she left, and was never heard of again. Even for a follow-up study a few years later, my group was unable to get in touch with her.

There is one other patient, whom I did not mention before, but whose story is worth recounting in the present context. She was a woman of 36, who had enrolled in a study for patients with medication-resistant auditory hallucinations that we carried out at the time in collaboration with Professor Iris Sommer from the University Medical Center Utrecht . In addition to the voices , our patient suffered from various additional symptoms and disorders, including two types of metamorphopsia . We later described her case in the scientific journal, *Psychopathology*, where we gave her the name of ‘patient A’, for ‘Alice’, and detailed how we had attempted to treat her voices with fMRI -guided repetitive transcranial magnetic stimulation (rTMS) , an experimental method that involved administering magnetic pulses to the brain with the aid of a figure-of-eight coil held over the head [8]. What rTMS does is to switch discrete groups of neurons either ‘on’ or ‘off,’ located at a depth of 1–2 cm under the skull. In our study, the location of treatment was determined in advance, using an MRI scanner that allowed us to visualise the parts of the brain that were active while patients were hearing voices. Thus, by requesting ‘patient A’ to press a balloon during scanning whenever she heard voices, and to release it when they abated, we had obtained activation maps of her brain that showed us exactly where that activity took place. By later aiming the rTMS coil at the location that had shown the most prominent activity (which in her case was the left temporoparietal junction) and treating her for 3 consecutive weeks, for 5 days a week, 20 minutes a day, not only the auditory hallucinations disappeared but, to our surprise, also the metamorphopsias and other visual symptoms that she had suffered from. In our paper, we hypothesised that this was either due to a placebo effect or due to a network effect. Regarding the latter possibility, we speculated that the part of the auditory network that we had treated with our magnetic pulses might have stood in direct connection with the visual network , parts

of which had subsequently also been ‘switched off’. Whatever the exact cause, the voices and other symptoms stayed away for 8 months, which was a huge relief to this severely ill woman. I remember clearly how burdened and weighed-down she seemed when she arrived for our first meeting, and how much difficulty she had had concentrating on our interview, searching for words, and being constantly distracted by her symptoms—and how vivid and bright she had been during our follow-up meetings post treatment. She had not felt so good for many years; she was able to pick up many activities she had been forced to neglect for many years, going out again, visiting friends, and generally living life the way she had before she had become ill. When—after 8 months—her symptoms had returned, patient A was back to the fragile and desperate state she had been in when we first met. She requested us to let her have yet another go at the rTMS . Although the study protocol did not allow for that, we gladly granted her wish and treated her for another 3 weeks—exactly the same as before, albeit outside the context of our study. Like the first time, the effect was dramatic and patient A parted our programme again symptom-free.

These are all single-case descriptions, which obviously cannot replace the clinical trials that should be carried out to assess the effects of these treatments in larger groups of patients to allow for the development of proper treatment protocols . Nevertheless, I mention them here, for what they are worth, because off-label treatment is all that is currently available for cases with no demonstrable aetiology, and, sometimes, the symptoms of Alice in Wonderland syndrome are so severe that we cannot simply sit and wait until science has picked up on this topic.

6.3 Future Directions

What should we ask scientists to do anyway, in order to advance the cause of people suffering from Alice in Wonderland syndrome? What would be needed to render this orphan disease its rightful place on the agenda of scientific committees, educational institutions, and insurance companies, and in the hearts and minds of researchers and clinicians? Although no hard data are available, there must be numerous people out there suffering from this virtually unexplored condition; they may not even know what it is called and, in some cases, not even realise what is wrong with their perception, or may have it misdiagnosed as something entirely different

(like schizophrenia or personality disorder) and perhaps receive treatments that may do more harm to them than good. As we saw, the condition is so obscure that only a minority of medical specialists would recognise it. By describing the syndrome and lending it this uniquely evocative name, Todd did a great service to all those people, worldwide, who suffer from perceptual distortions. And yet, after his seminal publication in 1955, neither he nor anyone else seems to have gone on to promote its general acceptance as a disease entity. If it had not been for those few scientists who kept on publishing papers throughout the years, Alice in Wonderland syndrome would probably have been forgotten by the medical world, left behind in the orphanage for medical curiosities, to live there quietly by itself, being of service to no one but the occasional clinician who was curious enough to look for a diagnosis beyond the official canon of medical diseases. I think that you will agree with me by now, that the syndrome deserves better than that.

Therefore, what is needed in the first place is an increased awareness of Alice in Wonderland syndrome. Everybody knows what the flu is, whereas hardly anyone knows what Alice in Wonderland syndrome is. That needs to be changed. That, obviously, is also the principal goal of the present book. People suffering from Alice in Wonderland syndrome need to be able to find their way to trained specialists. That starts with them being aware that there is a name for this whole bizarre collection of perceptual distortions that they may be suffering from, and also the availability of specialists with sufficient knowledge about how to interpret their stories and establish a proper diagnosis.

What is also needed is that international scientific committees recognise the syndrome as a disease category and work towards the development of official diagnostic criteria. As long as such criteria are not available, any clinician worth his salt will probably do just fine on the basis of good clinical practice, the way John Todd also managed without official criteria at the time. However, the inclusion of Alice in Wonderland syndrome in official diagnostic classifications would offer important benefits. Obviously, it would further general awareness of the condition; in addition, it would promote a uniform approach towards diagnosis, facilitate communication among clinicians *and* researchers and help to focus and homogenise scientific studies. On a more practical note, inclusion in official diagnostic classifications is often a prerequisite for reimbursement of medical costs by

insurance companies. Without a DSM or ICD code, many of those companies simply deny remuneration—which may become costly, especially when MRI scans and other auxiliary investigations are required to investigate the presence or absence of the underlying pathology.

Regarding scientific research, what is urgently needed are large-scale epidemiological studies to establish the prevalence rate of perceptual distortions in the general population and in populations at risk, such as those with migraine, epilepsy and substance abuse. On the basis of such studies, we will then be able to develop better cut-off values for what we consider ‘cases’ and ‘non-cases’. Moreover, they can help us to gain better insight into the conditions underlying Alice in Wonderland syndrome and, perhaps, also into protective factors. Such large-scale studies should preferably be complemented by genetic studies, aimed at unravelling hereditary patterns associated with an increased risk for developing perceptual distortions. In addition, one can think of numerous studies in the area of basic neurophysiology, aimed at elucidating the mechanisms underlying individual perceptual distortions, comparable to the way Hubel and Wiesel studied orientation columns in visual cortex. Importantly, such studies may now benefit from neuroimaging techniques that were not available during their time.

Regarding treatment, therapeutic algorithms need to be developed for the different conditions that may underlie Alice in Wonderland syndrome, and—especially—for cases where such an underlying condition cannot be demonstrated. To that end, studies are needed that assess the efficacy of medicines and other interventions, preferably with the aid of randomised, double-blind, placebo-controlled designs. Because the success of such studies depends on the inclusion of sufficient numbers of participants, they are best carried out in multicentre collaborative projects. Alternatively, one might consider creating an international database where findings from different institutions can be pooled, which increases the chance for sufficient statistical power. Obviously, that would require working with uniform criteria throughout, which would be yet another argument in favour of the development of official diagnostic criteria. Of special interest, in this context, would be the question whether substance-induced cases of Alice in Wonderland syndrome (i.e. HPPD) are really so much less amenable to treatment than others. If so, one might consider including that information in drug-awareness campaigns, aimed at preventing HPPD.

Last but not least, what deserves our special attention is the mechanism underlying ‘cerebral asthenopia’, the mysterious process which allows sufferers of Alice in Wonderland syndrome to function normally for a couple of minutes before their symptoms kick in.

In sum, there is a lot of work to be done in this area. Luckily, then, there is also a lot to be gained, not least for all those numerous people suffering from Alice in Wonderland syndrome who may not even know what they are suffering from.

References

1. [Anonymous] (1956) The “Alice in Wonderland” syndrome: relation to migraine. Based on an article by Todd J (1955) *Canad Med Assn J* 73: 701. In: Abbott Laboratories (ed) *What’s new*. Special Christmas Edition 1956. Abbott Laboratories, North Chicago, pp 22–24
2. Todd J (1955) The syndrome of Alice in Wonderland. *Can Med Assoc J* 73:701–704
[[PubMed](#)][[PubMedCentral](#)]
3. Lippman CW (1952) Certain hallucinations peculiar to migraine. *J Nerv Ment Dis* 116:346–351
[[Crossref](#)]
4. Todd J, Collins AD, Martin FRR, Dewhurst KE (1962) Mental symptoms due to insulinomata. Report on two cases. *Br Med J* 2(5308):828–831
[[Crossref](#)]
5. World Health Organisation (1992) *The international classification of diseases, 10th revision*. World Health Organisation, Geneva
6. American Psychiatric Association (2013) *Diagnostic and statistical manual of mental disorders, 5th edn*. American Psychiatric Association, Washington
[[Crossref](#)]
7. Morehead DB (1997) Exacerbation of hallucinogen-persisting perception disorder with risperidone. *J Clin Psychopharmacol* 17:327–328
[[Crossref](#)]
8. Blom JD, Looijestijn J, Goekoop R, Diederens KJM, Rijkaart A-M, Slotema CW, Sommer IEC (2011) Treatment of Alice in Wonderland syndrome and verbal auditory hallucinations using repetitive transcranial magnetic stimulation. A case report with fMRI findings. *Psychopathology* 44:337–344
[[Crossref](#)]

Footnotes

1 For implementation in the ICD or DSM , these classification systems might benefit from a new chapter called Perceptual Syndromes, in which not only Alice in Wonderland syndrome finds its rightful place but preferably also conditions such as musical hallucinations , sexual hallucinations , tinnitus , exploding head syndrome , clinical zoanthropy and the incubus phenomenon , all of which are currently underserved in these systems.

2 Thus, I myself had a patient who suffered from visual *and* auditory distortions, the latter consisting of an inability to filter environmental sounds and to distinguish between ‘foreground’ and ‘background’ sounds. As a consequence, my patient perceived all sounds as equally loud and had great difficulty concentrating on the ones that were relevant. Although not mentioned by Todd in his original paper, this may imply that distortions experienced in the auditory modality should also be allowed to count as symptoms of Alice in Wonderland syndrome.

7. Did Charles Dodgson Suffer from Alice in Wonderland Syndrome?

Jan Dirk Blom^{1, 2, 3}

- (1) Parnassia Psychiatric Institute, The Hague, The Netherlands
- (2) Leiden University, Leiden, The Netherlands
- (3) University of Groningen, Groningen, The Netherlands

Congratulations! Having come this far, you now belong to an exclusive group of people who are up to date with Alice in Wonderland syndrome. You would probably never have guessed how easy it would be to become an expert on this medical topic—and yet, here you are, knowing more about it than the majority of medical specialists worldwide.

Nevertheless, a few mysteries still remain. So, let us start with the question that has been burning in our minds since the beginning of this book: Now that (more or less) all has been said and done, how much evidence do we have that Charles Dodgson actually experienced the perceptual symptoms he described so expertly in the *Alice* book?

The short answer to that question is that we have no evidence at all.

At least no direct evidence.

We simply do not know.

In his diaries, Dodgson never says anything like, ‘Had the most curious experience—while looking out over Tom Quad, saw everything as slanted’ or ‘Consulted the eminent alienist, Professor X, to ask his opinion about my legs shutting up like a telescope’. There is nothing of the kind, nothing even like that. Not once. The closest that we come to finding a description of anything that might hint at Alice in Wonderland syndrome in the diaries, are the passages where Dodgson refers to ‘seeing fortifications’—which are,

however, geometric visual hallucinations, not distortions—and the single passage (described earlier), where he reflects on his ‘foreknowledge’ of the Hymn that the curate gave out in Church. Although it is uncertain whether the latter experience was a *déjà* phenomenon, even if it had been, it would have been an *adjuvant* symptom of Alice in Wonderland syndrome and not a core symptom. In sum, I was unable to find any direct evidence, either in the primary or secondary sources, that Dodgson experienced perceptual distortions—not before and also not after he wrote the *Alice* book.¹

So, what about indirect evidence? If we zoom in on the one document that sparked this whole discussion, what does it say when someone casually describes 13 different symptoms of Alice in Wonderland syndrome in a single book? There we can read about illusory feelings of levitation, hyperschemata, hyposchemata, micropsia, macropsia, microsomatognosia, macrosomatognosia, somatopsychic duality, prosopometamorphopsia, loss of stereoscopic vision, time distortion, derealisation and depersonalisation. Moreover, all these symptoms have been so ingeniously incorporated in the *Alice* book (some of them multiple times), and each of them rendered in such a vivid way that we can totally imagine how it must be to experience them.

One might argue that this is a matter of circular reasoning: By first having Todd group these symptoms together by virtue of the book, and then saying, ‘Hey, look how many symptoms of Alice in Wonderland syndrome there are in the book’. However, since the majority of these symptoms had been described in the medical literature *before* Todd had linked them to the *Alice* book, this is certainly not a tautology. As a consequence, the real question is: *How great are the odds that a non-medical author would randomly describe these 13 different symptoms in a book written for the purpose of amusing and entertaining children; the symptoms themselves being so obscure that hardly any health professional knew about them at the time, and that it would take another 90 years before an extraordinarily gifted psychiatrist, named John Todd, would first think of describing them in connection with one another?*

I don’t think we need advanced statistics to establish that the odds of that happening tend to zero.

So, yes—on the basis of the descriptions in the *Alice* book alone—I believe that we possess overwhelming evidence that Dodgson had intimate knowledge of a large number of symptoms that we now consider part of the

Alice in Wonderland syndrome. It may seem like a detour to investigate so many other sources before we come back to the one source that set this quest in motion, but those other sources needed to be checked anyway in search of additional evidence. Having done that, and having found none, we can now safely conclude that the *Alice* book itself contains all the evidence we need.

Still, that does not prove that Dodgson actually experienced these phenomena himself. We now know that he incorporated many elements from real life in his book—but who can say that this was something he had also experienced himself? Therefore, the next question is: What might have inspired him to describe them? Had he perhaps heard about them from someone else? With his interest in medicine, and his eagerness to help and dispense medical advice, whether or not with the aid of his homeopathy kit, could it not be possible that he had stumbled upon one of those very rare individuals who had experienced not just one, but a whopping 13 different symptoms of Alice in Wonderland syndrome? Or several individuals who had each given him a description of a few of those symptoms? Could it have been his nephew, Stuart Dodgson Collingwood, for example, or Charles's own brother, Skeffington Dodgson (1836–1919), who were both known to suffer from epilepsy? Or any of the number of persons he had encountered over the years while they were actually having a seizure? Could they have told him afterwards? Or could it have been Alice Liddell? How touching it would be to imagine Dodgson learning about these peculiar perceptual phenomena from his beloved little muse, and then 'handing them back' to her in the context of a story that she loved, thus helping her to digest those haunting experiences and come to terms with them. How touching—and also how pathetic—since there is no indication whatsoever that Alice Liddell experienced perceptual distortions, let alone confided in Dodgson about them. Obviously, the same holds true for the other persons I mentioned: Which brings us back to the question as to whether Dodgson himself had been the one who had experienced them and, if so, what could have caused them.

Since several interesting hypotheses have been offered in the past, what I will do here is provide a re-examination of the existing evidence and, where evidence is lacking, simply ask myself aloud what might have been at play. That way, in the end, it will be up to you to weigh the evidence and conclude for yourself whether or not there is sufficient reason to believe

that Dodgson did indeed suffer from the syndrome that was named after his famous protagonist and what may have been the most likely cause of the related phenomena.

7.1 Health Myths

To investigate the nature and cause of Dodgson's alleged perceptual distortions, some authors mobilised solid medical expertise. Prominent among them are Goodacre [1], Podoll and Robinson [2, 3], and Hart [4]. Others, however, have not always been quite so scrupulous. Thus, throughout the years, much has been made of mere snippets of information about Dodgson's physical and mental health. In his own time, a rumour already circulated that he had become mentally ill. As Dodgson himself exclaimed (with barely hidden amusement) in his diary, after having made a new acquaintance who clearly had no clue as to who he was,

Only yesterday Mrs. Nash told me she had heard the author of Alice had gone mad! [5]

And that was only the beginning: Dodgson has since been called depressed, suicidal, a paedophile, a migraineur, an insomniac, an epileptic, an alcoholic, a cocaine addict, an opium addict, a cannabis addict and a magic-mushroom addict, to mention just a few of the more common allegations on offer. Others have pointed at his use of dangerous substances such as arsenic and sulphurous acid, although without mentioning that he took the arsenic in homeopathic dosages (implying that the solution was diluted to such an extent that not a single molecule of arsenic remained in it) and that sulphurous acid (a common disinfectant at the time) is something other than *sulphuric acid*, which is a very dangerous substance indeed. Moreover, Dodgson probably never used the sulphurous acid prescribed to him, since he disagreed with his physician's diagnosis and, therefore, preferred to try something homeopathic. Still others believe that Dodgson must have had difficulty keeping his fingers off the LSD—which would have been a truly remarkable feat, since this hallucinogenic substance was developed half a century after his death.

But what about those other possibilities? In a scholarly analysis of famous people who allegedly suffered from epilepsy, Hughes reveals that

Dodgson ‘was involved with cocaine’, although, unfortunately, without providing any sources to back up that claim [6]. On the Internet (not Wikipedia this time), I found another tantalising reference. The source mentioned was Lennon’s *The Life of Lewis Carroll* [7], which I subsequently binge-read in a single day—and guess what? Another dead end. Lennon’s classic was a marvellous read, but nowhere does it mention the use of cocaine or in fact any psychoactive substance, other than alcohol. The only reference to cocaine that I found (as detailed in Chap. 4) was its one-time use as a local anaesthetic to facilitate a minor surgical procedure [8]. Since cocaine under such circumstances does nothing but numb the peripheral nerve endings in the patch of skin to be incised, it requires a considerable stretch of the imagination to conclude that it has any influence on the central nervous system, let alone that it is the source of an addiction. Opium (also mentioned by Hughes [6]) was frequently employed in 19th-Century medicine in the form of laudanum , a tincture of 10% opium, equivalent to 1% morphine . It might perhaps have been prescribed to Dodgson during one of his many disease periods, or even to alleviate a toothache, since it was distributed liberally at the time to treat almost any ailment imaginable. It is pure speculation, however, as to whether he ever used it, since there are no known records to confirm this. For the use of cannabis (also suggested by Hughes [6]), the same holds true. As regard the alleged use of magic mushrooms , it was Carmichael who suggested that Dodgson had either read about the hallucinogenic effects of *Amanita muscaria* or experimented with this classic red-and-white mushroom himself—although, here also, without providing any evidence that he actually did [9].

So what about alcohol ? Could that have been the source of the mind-bending perceptual experiences described so vividly in the *Alice* book? Such distortions may accompany not only delirium tremens but also alcohol hallucinosis , alcohol withdrawal and even ordinary alcohol intoxication . As a curator of the Common Room at Christ Church , Dodgson had access to sufficient quantities of alcohol to put the entire academic staff at Oxford into a coma—buying wine and sherry in bulk and sampling the merchandise on behalf of his confreres, turning himself into a self-professed wine expert in the process [10]. Since he also had a habit of skipping midday meals, swapping them more than once for a glass of sherry and carrying a flask of his own preferred brand with him when he dined out, we may ask ourselves

whether alcohol in fact played a more prominent role in Dodgson's life than has hitherto been acknowledged [11]. However, all sources indicate that he used alcohol in moderate quantities, drinking no more than one or two glasses of sherry, wine, ale or beer per day and only occasionally a few glasses more. If that should qualify as an alcohol addiction, then the French should probably be considered collectively addicted. That said, we all know that alcoholics who successfully manage their drinking behaviour in accordance with their social and occupational schedules may go undetected by their surroundings for astonishingly long periods of time. Since Dodgson was a master at orchestrating his life and extremely picky about the topics he chose to report in his diary, we cannot be entirely sure whether, or not, he was secretly addicted. Even though there is no evidence for it, let us place a question mark here as a reminder that, in any given situation, the real problem may always turn out to be alcohol, lest we forget about it when we are up to our necks in discussing other possibilities.

To see what other possibilities there are, let us now revisit Dodgson's medical history and connect it with what we have learned in the meantime about Alice in Wonderland syndrome.

7.2 Dodgson's Medical History Revisited

One of the myths created by Dodgson was that he had always been thoroughly healthy. That is how he wanted the world to see him and that is probably why (in his letters and diaries) he proclaimed time and again that his health was 'something to be most thankful for'—even though he suffered deeply from his speech and hearing impairments, and had to cope with painful conditions ranging from neuralgia, headaches and lumbago to annually recurring infectious diseases that often kept him on the sofa for weeks on end. He, for whom language was so vitally important, was deprived of the capacity to hear and speak freely, and he, who was so fond of exercising and long walks, was literally benched for long stretches of time due to his recurring 'synovitis'. On top of that, his face blindness turned his social life into a recurring nightmare, undermined as it already was by his speech and hearing problems. What was therefore probably true about Dodgson's claim to health is that he had found ways to cope with his numerous health problems without succumbing to depression or despair—

which is indeed something to be thankful for even though it is not quite the same as *being* healthy.

Since we already have an overview of all that is known about the medical conditions Dodgson suffered from (Appendix A, Table A.1), I will focus here on several aspects that might help us to find an answer to the questions that remain. First of all, let us see whether there are any indications for psychological or psychiatric conditions in Dodgson's medical history that might justify the assumption that he experienced perceptual distortions himself. Since we already know that Alice in Wonderland syndrome is a thoroughly neurological affair, this may not seem the most obvious place to start; but, then again, perceptual distortions have also been described in the context of psychiatric disease, so this is a topic that we cannot just simply ignore. Moreover, so much has been said about Dodgson's mental health in the past that it gives me a welcome opportunity to deflate a few more myths while we are at it.

7.2.1 Dodgson's Psychological Profile

Every health professional knows that one cannot carry out a reliable psychiatric assessment without having seen and examined someone in person. However, with that proviso firmly in mind, I think we can safely conclude from the wealth of primary sources that Dodgson never suffered from a depressive disorder, or in fact from any other major psychiatric disorder—as we know them today, or as they had been known back then, during the second half of the 19th Century. He certainly was not 'mad', as Mrs. Nash had offered so unceremoniously. More specifically, there is no proof that he ever suffered from a psychotic disorder, a mood disorder, dementia or anything else that we categorise as 'severe' in my discipline. Undoubtedly, he had gloomy thoughts from time to time and grieved after the death of his mother and, especially, of his father; meanwhile, his diaries also show that, for a long time, he was plagued by recurring feelings of guilt and self-doubt, possibly mostly during the years spanning 1862–1866, about which we know so little. And yet, nowhere do we find any indication that he was depressed for weeks on end and that during such times of gloomy thoughts he lost interest in his activities, withdrew from society or wasted away due to a lack of appetite and sleep—all of which are cardinal symptoms of major depressive disorder. Even in the passages that show his darkest moments of self-recrimination, his diary allows us only brief

glimpses of his despair, while, in between, it describes his life as going on uninterruptedly, apparently unaffected by whatever it was that haunted him. His diaries and letters, and even his poems, show that he did indeed think a lot about death but not in any way that we can construe retrospectively as suicidal ideation. So, whatever an actual psychiatrist might have brought to light at the time (at whatever point of his life, I am inclined to add, although we cannot be sure about the 4-year gap), it would probably have fallen outside the domain of major psychiatric disease. That includes the so-called split or dual personality that biographers ever since Langford Reed have offered as an explanation for his eccentric behaviour—as if ‘Lewis Carroll’ had been split off from the actual person called ‘Charles Dodgson’, the two of them functioning more or less independently of each other and dominating his actions in turns [11]. He was, undoubtedly, capable of compartmentalisation, meaning that he had the ability to adapt his behaviour and communication style to the social circumstances, allowing him to act like the don and gentleman that he was in academic circles, to adopt the role of a bohemian among his artist friends and to sit under the table with his child friends if that was what the game required. Meanwhile, he had also been well capable of giving and withholding information as he saw fit: Posing as a nonconformist in religious circles, as an open-minded spiritualist among his fellow members of the *Society for Psychical Research*, and as a boring mathematician (he admitted that to himself) among his students. In all that has been written about him, we find no indication whatsoever that this caused him any confusion over who he was, over what he had done while assuming a different role, or over the way he was supposed to act under all those different circumstances—except, perhaps, when an unexpected role reversal was required, such as when the telling of a story to his child friends was interrupted by the arrival of an adult. Therefore, I would tend to argue that Dodgson seems to have mastered an unusual set of social skills and an unusual capacity of juggling those around in varying social contexts rather than having suffered from dissociative identity disorder (as the ‘dual personality’ is called today).

In sum, I do not believe that there is sufficient evidence to substantiate the claim that Dodgson suffered from any type of major psychiatric disease. That cannot be the explanation we are looking for. So, what about ‘minor’ illnesses? After all, psychiatry is not only about raving madness, so we

should certainly not forget about the simpler afflictions that one can suffer from.

Thus, continuing our search outside the domain of major psychopathology, it may be interesting to have a look at two descriptions of ‘character’, as drawn up by contemporaries of Dodgson’s . At 20 years of age, he had had his character ‘read’ in Edinburgh by the phrenologist Edward Hamilton , who had ‘felt Dodgson’s bumps’ and subsequently declared that:

This gentleman has eight very prominent traits in his Character, namely, a strong love of children; a strong love of friends; much emulousness and amiability; much Circumspection; Lofty generous sentiments; much good taste for order & dress & elegance; Excellent analogical reason; & deep penetrating causality to trace the relation between cause and effect... [12]

Even though phrenology had gone out of vogue in Edinburgh around 1840, ‘having one’s bumps felt’ was apparently something that people still did from time to time, whether or not for entertainment. Another description stems from a woman who claimed to be a clairvoyant , possibly Maria Katherine Anderson (d. 1889), whom Dodgson had met (by invitation of a Dr. Erskine) at the Deanery. By holding in her hand a folded piece of paper containing some words written by persons unknown to her, the woman described the characters of those present, saying about Dodgson:

Very clever head; a great deal of number; a great deal of imitation; he would make a good actor; diffident; rather shy in general society; comes out in the home circle; rather obstinate; very clever; a great deal of concentration; very affectionate; a great deal of wit and humour; not much eventuality (or memory of events); fond of deep reading; imaginative, fond of reading poetry; may compose. [11]

Both readings could probably have been performed after half an hour in Dodgson’s company, provided that he could have been persuaded to talk about his favourite pastimes—so perhaps, it is not a surprise that both, on the basis of what we know about him, appeared to be fairly accurate. To this, we may add, without the need for any psychological testing, that Dodgson possessed several obsessive and compulsive character traits. The

way he dressed, put on his gloves, cared for his teeth, prepared his tea, kept his diary, indexed his entries, archived his letters and photographs and carefully listed the names of pretty girls whom he had met (complete with dates and all), speaks for itself in this respect. We might even say that his obsessive traits sometimes bordered on fetishism, considering his habit of photographing people, asking for their autographs and visiting cards and collecting tiny locks of hair from his child friends. I somewhat reluctantly use the term fetishism here because we already saw that the photographs also served aesthetic purposes and, moreover, helped him to keep track of who was who in his extensive social network predating Facebook—hindered, as he was, by his face blindness. As a consequence, collecting autographs and visiting cards may also have served to document whom he had met. And yet, there also appears to have been a narcissistic side to this, as he only collected them from people he considered ‘up there’ with him in his social class, or preferably ‘above’ him. Dodgson had a very strong class consciousness, which led him to seek the acquaintance of professors, clergymen, artists, nobility and—if he got the chance—Royalty, hunting them down and literally stalking them, the way he had done, for example, with Alfred, Lord Tennyson (1809–1892), the Poet Laureate, whom he had sought to approach via various family members, and whom he subsequently followed to his holiday destination to lie in wait for him, day after day, until he saw a chance to make his acquaintance. Upon completion, he would write about such ‘lion-hunting’ expeditions in his diary or to his sisters, boasting about his latest successes, which had often been accomplished with the aid of his faithful camera, which helped him to gain access to people who wished to be photographed and would otherwise have been ‘out of his league’.² The reason for collecting locks of hair from his child friends can hardly have served any other purpose than the desire to possess something tangible of them—something to be taken out of a drawer once in a while and looked at, touched and smelled, perhaps, to remind him of the bond that he had created with this particular child by taking something that was hers in a most intimate way.³ Whether collections such as these—notably the photographs and the locks of hair—also had a sexual connotation for him, is impossible to know. Moreover, it is so sensitive an issue that I gladly leave this as a question to be answered by others—if they can. Speaking of which...

7.2.2 Sexuality and Sexual Characteristics

We already saw that a great deal of speculation has taken place as to whether Dodgson had been asexual or not, whether he had been capable of romantic love (and, perhaps, had been rejected), and even whether he might have led a life full of secret sexual encounters. Considering the way in which he carefully filtered what went into the diary, plus his awareness that it might be read by others, *and* the obvious realisation that his fellow Victorians would not take lightly to any hint or suggestion that he fancied the children or women with whom he had surrounded himself, it cannot be ruled out with certainty that he had been a Don Juan in disguise. Still, in that case, Dodgson must have been very skilled at hiding his frivolous escapades—if they ever took place—with all those parents keeping a watchful eye out over their daughters, prepubescent or not, and all the gossip that went on around him anyway. Moreover, it is hard to imagine that none of his former lovers—if they ever existed—would not have gone on to assert that they were the mother of an illegitimate child of his, if only to lay claim to the considerable fortune that everyone thought he had accrued.⁴

Whereas the possibility of an active sex life—however unlikely—can therefore not be totally dismissed, to make a case for asexuality may not be as desperate as it sounds. What if Dodgson had sought the intimate company of girls and young women, and enjoyed the exclusivity of being in their presence without experiencing any marked sexual or romantic overtones? Or, if he had experienced them, had enjoyed them in a platonic way? Wouldn't that be possible?

As indicated before, asexuality is extremely rare in adults. And yet, it is not unthinkable that Dodgson belonged to the exclusive group of male adults who are devoid of any sex drive. This is another mystery that must go unsolved, although there is something more to be said about it that may throw some light on his ways with women.

As recounted before, at the age of 17, Dodgson had suffered from mumps, an infectious disease that can be complicated by meningitis, pancreatitis, deafness and—perhaps significant in the present context—orchitis or testicular inflammation. It is very likely that his pre-existing hearing impairment was aggravated by the mumps, leaving him deaf in the right ear. Whether he also went on to develop orchitis is unknown. Prepubescent boys suffering from mumps rarely go on to develop testicular complications, but in adult men, it is the most common type of

complication, affecting about 5 to 37% of them [15]. At the age of 17, Dodgson could no longer have been considered a prepubescent boy and, therefore, belonged to the group at risk for orchitis .

What happens in such cases is that the mumps virus targets the testicles directly within the first few days of infection, destroying the parenchyma (the basic cellular tissue the testes are made of), thus causing a loss of function of one or both testicles. Initially, this causes pain, a ‘heavy’ feeling and a tender, swollen, red or purple aspect of the scrotum, along with nausea, vomiting and a further increase of the fever already caused by the mumps. Urination may become painful, and, in sexually active men, the sperm may become tinged with blood. Orchitis is lethal in a minority of cases, but those who survive it may find that one or both testicles have shrunk and that they no longer perform their proper functions. Apart from their role in the production of sperm cells, the testicles have a role in the synthesis of androgens, the hormones responsible for the development of secondary sexual characteristics. As a consequence of mumps orchitis, levels of testosterone and other androgens tend to plummet to unusually low levels, which happens in 40–70% of the cases, most often unilaterally, and in 15–30% bilaterally [16]. In the latter case, 13% of the patients go on to develop testicular atrophy (a shrinking of the testicles), a reduced production of sperm cells (oligospermia) and hence a strong decrease of their fertility—or even infertility .

Additional consequences of such a dramatic decrease in testosterone levels may be a loss of libido, impotence , gynaecomastia (i.e. the development of female breast tissue) and a failure to develop typically male characteristics such as heavy muscle and bone mass, body hair and low voice. Thus, an adolescent male affected by bilateral orchitis basically faces infectious castration . Being unable to go on developing typically male physical characteristics, he will then reach adulthood with a relatively low body mass, a frail habitus, feminine facial features, a minimum of facial hair and body hair (leaving the hair of the head unaffected), a high-pitched voice, little or no sexual drive , and sometimes also impotence, infertility and female breast tissue. Photographs of Dodgson indicate that the latter was not the case—although, for the record, it should be noted that one can never rule out the presence of breast tissue without a proper medical examination.⁵ So here is another question mark to be placed, and to be held firmly in place, since we really do not know whether or not Dodgson had

any female breast tissue. As regards the other consequences of mumps - related orchitis , they would seem to apply remarkably well in his case, including the high-pitched voice, the frailty, the soft facial features and the —possible—asexuality .

Thinking this through, this might go a long way in explaining why Dodgson might have envied the Dean's place in the Liddell family. If this is what had actually happened to him, with the mumps-induced orchitis and the subsequent detrimental effects on his sexual drive , he might have been physically incapable of starting a family. Having grown up in a lively household with ten siblings, and being such an obvious family man, with his love for children and female company, this must have posed a huge existential problem to him, even without the need for an active sex life. And now, having been introduced into the Liddell household, with those children of unsurpassed beauty and wit, a wonderful mother, and that outdoorsy husband of hers who so conveniently left a vacant spot to fill—which, moreover, he was allowed to fill—would it not have been possible that he had at least fantasised or dreamed sometimes, however briefly, of taking the Dean's place for real? Dodgson was a rational person, who was too much aware of the social conventions of his day to ever think that this would have stood a chance in actual practice—however, having already ended up in this wildly unconventional situation, wouldn't it have been the next logical step for him to daydream about? Again, these are a lot of 'ifs'. So let us place another large question mark here and face the fact that we will probably never know for sure whether any of this took place.

What we do know is that it would have been unthinkable, especially in the Victorian era, that anyone outside the immediate family circle would have been told about so sensitive an issue. As a consequence, our chances of finding any confirmation of mumps -related orchitis and/or asexuality in letters or other written sources is negligible. This leaves us no other option than to tread extremely lightly here and conclude that we can by no means be sure whether Dodgson had been asexual, even though (with hindsight) he appears to have fit the bill rather well. On the basis of the odds mentioned above, the chances that he had suffered from unusually low androgen levels due to mumps-induced orchitis and, as a result, from a diminished sexual drive , lie somewhere between 1% and 10%, whereas the chances that he suffered from bilateral orchitis, and hence from total impotence and infertility , in the range of a tenth to 1%. While the odds are

thus stacked against this possibility, we may then ask ourselves how many adult men end up with a physique like Dodgson's, an all-consuming interest in little girls, and a marked predilection for children's books and games.

7.2.3 The Asperger Hypothesis

The latter remark may serve to remind us of what we already know, namely, that at least some of Dodgson's interests fell conspicuously outside the norm. Although certainly not all grown-up men are interested in sports, cars and women, they are an overwhelming majority in comparison with those who have an interest in wire puzzles, Euclid's theorems, time zones, occultism, devices that enable one to write in the dark, backgammon, acrostics, poetry, photography, literature, art, cathedrals, homeopathy, fairy stories and prepubescent girls. It is this idiosyncratic combination of interests that has prompted some authors to suggest that Dodgson may well have had a condition not yet envisaged during his time, called Asperger's syndrome—which is basically a fancy way of saying that he had peculiar interests and habits [17].

Asperger's syndrome can perhaps be understood best as a diluted form of autism or 'autism light'. Similar to people with autism proper, those diagnosed with Asperger's do not easily fit in with society, although they may sometimes surprise friend and foe by finding a niche in life where they can flourish and end up with numerous social contacts. As we saw, that was certainly the case with Dodgson. Not only had he found his niche as a tutor of mathematics, he went on to excel at what he did, publishing mathematical papers in *Nature* (which indicates that at least some of his scientific work was top-notch), writing poetry, becoming a pioneer of the fledgling discipline of photography, mingling successfully in artistic circles, producing a steady stream of literary output (with the *Alice* book as an unsurpassed highpoint), earning enough money in the process to sustain his large extended family (as well as numerous others), and building an extensive network of meaningful social contacts. Meanwhile, however, he remained something of a fish out of water, looked upon as an anachronism among the anachronisms that already populated Christ Church, dressing and behaving in his oddly stiff way, going against the grain over issues that others found trifling, consulting top medical experts and then discarding their opinion in favour of his own, developing grand designs for ideas that were at least slightly out of touch with reality, taking things either very

literally or mocking them in ways that ridiculed those who had brought them up, attaching more meaning to punctuality than is socially convenient and, generally, showing himself inflexible by imposing his own ideas upon others, and being unable to achieve more than a minuscule bit of change. In contrast to the majority of people with diagnosed autism, however, he did have a theory of mind; this implies that he was well capable of putting himself in someone else's shoes—especially those of children, who felt understood by him with unsurpassed subtlety and warmth. In addition, he was obviously well capable of social interaction with adults, despite the various practical obstacles he had to face.

Incidentally, these character traits—whether or not they would have justified, in our time, a diagnosis of Asperger's syndrome—also helped Dodgson to deal with the constant assaults on his physical health. By stubbornly refusing to let himself be turned into a cripple despite his recurring knee problem and lumbago, and by securing vaccinations against smallpox, preferring homeopathic medications over the often dangerous treatments offered by regular 19th-Century medicine, continuing year-in-year-out with his speech therapy, protecting himself against cholera and other diseases lurking in the drinking water and, in general, by continuing his private medical studies, he probably helped himself a great deal while battling his many ailments and preventing numerous others.

In other words, if we would conclude from this that the Asperger tag were indeed applicable as a retrospective diagnosis, then Dodgson must have belonged to a high-functioning subgroup who knew the sweet and the sour of this condition. Incidentally, in addition to the possibilities already mentioned, it may also have had some influence on his romantic and sexual interests, since asexuality, however rare, has been reported to be slightly more prevalent in the context of Asperger's syndrome [18]. Nevertheless, there is no reason why this condition—or any of the other psychological traits discussed so far—would have prompted him to experience perceptual distortions.

7.2.4 The Migraine Hypothesis

What may have caused them, however, as mentioned by several eminent authors, is migraine—a theme definitely worth revisiting. On the basis of Dodgson's own descriptions of 'fortifications', I agree that he must have experienced at least five instances of migraine, whether or not of the

classical type that goes with a headache . However, all these descriptions stem from a time *after* the *Alice* book had been written. What has remained a mystery to this day is the reason why Dodgson consulted the oculist, Dr. Bowman , in 1856, that is, 6 years before he started writing the book. Podoll and Robinson appeared to be relatively confident that the reason had been a visual manifestation of migraine [2, 3]. Interestingly, to back up their claim, in an article in *The Lancet*, they presented a drawing made by Dodgson between 1855 and 1862 (so probably before he had told the story to Alice and her sisters) that shows a remarkable blank space (Fig. 7.1) [2]. In it, we see a bearded, elf-like figure who is holding up his left arm. In the upper right-hand corner of the drawing, the left arm ends abruptly with the beginnings of a hand, which, for the rest, is incomplete. Likewise, it can be seen that the left side of the figure's head (also in the upper right-hand corner of the drawing) is missing. By comparing this drawing with another one, made by a migraine patient who deliberately left out part of a face to illustrate the 'rounded border effect' she had experienced due to a negative scotoma , Podoll and Robinson suggested that Dodgson probably suffered from the same condition and had, therefore, left the space blank without even noticing it.



Fig. 7.1 Frontispiece for *Mischmasch*, drawing by Charles Dodgson's (ca. 1855–1862)

It is a creative hypothesis. That said, we may ask ourselves whether this was the real reason why Dodgson had left his drawing unfinished. Had he worked in a fixed position without ever moving his head, and had he indeed experienced a negative scotoma at the time, then it might have been possible that he failed to notice the unfinished space in the upper right-hand corner. However, it is hard to imagine any artist to be working in such a fixed position, as if strapped onto a headrest or connected to an external fixation device. As this was obviously not the case, I find it difficult to believe that Dodgson never noticed—while moving his head or looking at his work from a different angle—that something was missing. Moreover, since negative scotomas tend to last no longer than just a few minutes, he would have become aware of his omission soon enough and then, probably, have finished what he had begun.

Therefore, I believe that it is much more likely that Dodgson left this particular space blank with the intention of adding an object (such as a treasure chest or an umbrella, or whatever object he may have had in mind), and that he either went on to some different project without finishing it or simply gave up because he had no idea how to make it work. After all, he had claimed never to have had a drawing lesson in his life and, in his diaries, complained repeatedly about his lack of artistic talents.

As a consequence, I still consider the nature of Dodgson's eye problem in 1856 to be a mystery and believe that the most likely explanation for Dr. Bowman's advice was not that this respectable specialist failed to establish a proper diagnosis, but that he considered the problem to be of a mental—rather than of an ophthalmologic or neurological—nature. Having in all likelihood been unfamiliar with the visual manifestations of migraine, Bowman may of course have mistaken a negative scotoma (had that been the problem) for a mental problem. However, since there is no way of knowing this, we fall short of any means to support Podoll and Robinson's tantalising claim that Dodgson suffered from migraine *before* he had written *Alice's Adventures in Wonderland*—nor, even, to substantiate the claim that he had suffered from a negative scotoma before that time, which was the cornerstone of their migraine hypothesis.

7.2.5 The Epilepsy Hypothesis

As we saw, the other major hypothesis, the one involving epilepsy, had been unlikely from the outset. However, there is more to be said about the

reasons why it is so unlikely.

First, it is worth noting that epilepsy has had opposing metaphysical connotations since times immemorial. On the one hand, it has always been associated with inferiority and decline, as exemplified, in Dodgson's time, by the 19th-Century degeneration theory, which passed it off as an example of the backwards evolution that society had allegedly fallen prey to [19]. Thus, people suffering from epilepsy were looked upon as second-rate citizens, less advanced than their fellow-citizens and probably guilty of bringing about their own condition, or else having to thank their forebears for it, who had undoubtedly done something bad (in a physical, psychological or moral sense) and passed that 'something' on to their offspring. Because of these connotations, the Dodgson family may not have been too eager to let the epilepsy story out of the box and may even have sought to suppress Charles's own stories about it. For he himself did not appear to be at all taken aback about his alleged seizures. On the contrary: He spoke and wrote freely about them and seemed only too glad to be able to share his experiences with others. The reason for that may have been that he had a more neutral stance towards epilepsy, due to his biomedical orientation, or—taking this argument one step further—because he may have associated it with something worth pursuing. Contrary to the way epilepsy was characterised by the 19th-Century degeneration theory, it has of old also been labelled as a mystic event, during which the person 'stricken' is allowed to experience a revelation or a sense of unity with a higher power. Not for nothing did the ancient Greeks call it 'the sacred disease'. Followers of Hippocrates already sought to debunk the disorder's mystical connotations by trading the old name for 'the disease called sacred'; nevertheless, associations between epilepsy and the mystical have never gone completely out of fashion. It is not unthinkable, therefore, that the mere idea of having suffered an epileptic seizure—inside the Cathedral, no less, the most sacred place in Dodgson's immediate vicinity—may have seemed enticing rather than horrific to him, fascinated as he was by the paranormal anyway. Could that have been the reason why he wanted to believe so badly that his attacks had been epileptic in nature, clinging on to the idea with ever more determination each time he wrote about it, even in the absence of any new evidence?

The latter question raises the issue of what kind of evidence we would have wanted to see anyway, to help Dodgson make his case. As I explained,

the 19th-Century notion of ‘epilepsy’ was operationalised differently than it is today. In Dodgson’s time, the condition was primarily recognised (as it had been since Antiquity) by the presence of tonic-clonic seizures, meaning that the person suffering from it fell down, lost consciousness and showed a stiffening of the muscles (called the tonic phase), followed by an episode of rapid, rhythmic jerks of the arms and legs (the clonic phase). During the 19th-Century, it was already suspected that these seizures had something to do with ‘electricity in the brain’. The characteristic loss of consciousness, however, was attributed to spasms of the cerebral arteries, evoked by processes in the upper part of the brainstem. Only full-blown tonic-clonic seizures *with* loss of consciousness were considered ‘genuine’ epilepsy [20]. It was not until 1924 that the German psychiatrist Hans Berger (1873–1941) recorded the first human EEG, thus enabling the measurement and visualisation of changes in electrical currents in the brain’s cortical layers and the identification of epilepsy by its characteristic electrophysiological pattern. Ever since, the diagnosis of epilepsy has depended on the presence of such a pattern on the EEG rather than the presence of readily observable symptoms. As a consequence, it is now considered virtually impossible to prove epilepsy in the absence of positive findings on an EEG. Thus, while making a retrospective diagnosis is a tricky thing to do anyway, the absence of an EEG tracing makes it impossible for us to establish with any certainty whether Dodgson had really suffered an epileptic seizure or not, even if an account of a tonic-clonic seizure had been reported by one or more reliable eyewitnesses—which, moreover, is missing here. By Dodgson’s own account, neither the tutors, nor the verger, nor anyone else had heard him scream (which frequently occurs at the beginning of a tonic-clonic seizure) or witnessed him fall or shake his arms and legs. What is more, Dodgson appears to have been perfectly aware of the whole sermon, having followed it unhindered up until the point that he lost consciousness. From this, we may conclude that there had been no retrograde amnesia, and no aura (or ‘warning sign’).⁶ Another thing that also makes a seizure unlikely, is the absence of any postictal confusion. The way Dodgson described the situation, he apparently woke up asking himself why his pillow was so hard, but soon thereafter had been wide awake and clear-minded. People coming out of a tonic-clonic seizure, however, tend to be drowsy, disoriented and confused, sometimes for hours or even days on end. In addition, they usually have

sore muscles due to their fall and to the breakdown of muscle tissue stemming from the tonic and clonic phases. There is no indication whatsoever that Dodgson experienced any of this. Nor does he tell us that he had bitten his tongue or lost urine, as may be the case during seizures. Finally, as pointed out by Goodacre, 55 years is an unusually ripe age for a debut of epilepsy, especially in the absence of any underlying diseases such as a stroke or a brain tumour [1].

Therefore, in his paper on Dodgson's medical history, Goodacre hypothesised that Dodgson's 'attack' may well have been a case of carotid-sinus syncope, which basically means that he may have fainted due to pressure exerted on the vessels in the neck—probably brought about by the high collar of his frock coat, and possibly promoted by stress, sleeplessness and an empty stomach [1]. An alternative possibility, also suggested by Goodacre, is that Dodgson had either suffered a vasovagal collapse (i.e. 'simple fainting') or a transient ischemic attack (TIA). Although I do agree with Goodacre that epilepsy would have been an unlikely cause of Dodgson's 'attack', a TIA, in my opinion, would have been even more unlikely.

A TIA is a relatively brief episode of dysfunctioning of a circumscribed part of the brain due to a temporary lack of oxygen. Thus, that lack of oxygen does not affect the brain as a whole, as in cases of suffocation or cardiac arrest, but rather a discrete region that depends on a single artery or arteriole in the head. Typical causes of TIAs are blood clots that occlude such a vessel. Due to the ensuing lack of oxygenation 'upstream', the affected brain area fails to fulfil its function, thus causing symptoms such as temporary blindness in one eye, temporary speech problems and/or temporary paralysis of a leg and/or arm, to name some of the most common possibilities. The reason why such incidents are called 'TIA' rather than 'infarction' is that the neurons that make up the affected brain area remain intact—meaning that cell death does not occur because the problem is resolved before any structural damage sets in. The window for that to happen lies somewhere around 24 hours, hence the definition of a TIA as an ischemic attack that recovers within 24 hours.

As we saw, Dodgson did recover within that time frame, so in that sense he fulfilled at least one of the diagnostic criteria for a TIA. However, as TIAs usually involve the occlusion of a single artery or arteriole, thus affecting a relatively small brain area, located, moreover, within either of

the two hemispheres, and loss of consciousness requires both hemispheres to be affected, it is unlikely that Dodgson's 'attack' was caused by a TIA. Moreover, he never reported any of the typical signs of a TIA as outlined above. Therefore, and taking into account his attempt to get up from a kneeling position prior to the 'attack', a much more likely candidate for his loss of consciousness would be orthostatic hypotension, meaning that his blood pressure failed to rise to a sufficiently high level at the moment he tried to get up from the hassock, thereby temporarily depriving his whole brain of oxygen. It is a very common thing to happen, especially in the elderly, and certainly in combination with stress, sleeplessness and an empty stomach—as pointed out by Goodacre [1].

However, a salient detail that also needs our attention is that a mere 5 days before the incident, Dodgson had been laid up on his sofa again with yet another bout of 'synovitis'. The knee had been so much out of order that he had painted it with iodine and bandaged it, the way he had done so many times before [21]. How long he had been thus incapacitated is unknown but, considering the usual protracted duration of his 'synovitic' attacks, not all the swelling may have been gone when he resolved to go to church on that Sunday morning of 6 February 1891. Kneeling must have been painful under these circumstances, let alone getting up. Besides, sitting with bent legs is rather uncomfortable with a swollen knee and may cause numbness in the leg within minutes. Taking these circumstances into account, the cause of Dodgson's incident may well have been his swollen knee, and/or a numb leg, buckling away under his weight at the moment he attempted to rise up from his kneeling position—or else producing such a stab of pain that he may have fainted—in either case bringing him down with his head against the hassock. Another salient detail is that Dodgson was exasperated with the size of hassocks anyway, and that at some point he had angrily expressed his wish that a larger one be made for him to kneel upon [14]. It is a rather prosaic explanation for what may have happened on that memorable day in the Cathedral, stripped of any metaphysical overtones but, in view of the limited amount of information that we have, would this not be the most plausible reconstruction of what had happened? That said, epilepsy remains a possibility, of course, however unlikely it may currently seem. So, let us keep this one in mind, too.

7.2.6 Fever

And then there was the fever —the endlessly recurring fever, from his childhood years to (literally) the last day of his life. If anything qualifies for having produced perceptual distortions, in my opinion, this is it. Perhaps I am prejudiced because of my personal experience with the large hands during that bout of fever in Thailand, not to mention the experiences of people in my surroundings, including my daughter Esther, but even apart from that, I believe that the odds are in favour of the fever.

As one glance at Table A.1 (Appendix A) indicates, infectious diseases were the most frequent, and perhaps also the most incapacitating ailments that Dodgson endured throughout his life. What is more, he experienced them on numerous occasions *before* he wrote the *Alice* book. Seeing him now, with hindsight, being turned deaf in one ear by childhood fever and mumps, coughing out his lungs for months on end while battling pertussis, enduring several local infections (including bladder infection, so rare in men) and being held prisoner by his annually recurring bouts of flu, now complicated by bronchitis, then by his mysterious ‘ague’ and finally dying of (in all likelihood) pneumonia, it is safe to say that infectious diseases had given him a raw deal, even if for now we disregard the possibility that the mumps may have left his body frozen in a kind of adolescent limbo for the remainder of his life.

So, it is not just my own experience that prompts me to paint fever as the primary suspect. What is more, since influenza is still so prevalent today and most of us are only too familiar with it, it is easily overlooked how burdening this so-called common-and-garden disease can be and how little is needed to let it spin out of control. You may not realise it, but in 2017, worldwide, between 291,000 and 646,000 people *died* from seasonal influenza-related respiratory illnesses [22]. I am not talking here about the Spanish flu, which near the end of World War I wiped out millions of people. What I am talking about is ordinary influenza, of the kind that you have probably suffered from as often as I have. We all know about the grave threat posed by the Tropical disease, malaria, and the joint efforts of laboratories all over the world to find a cure or vaccine for it; however, with an annual global death toll of 445,000 [23], malaria and influenza-related diseases are in the same league. Influenza is a nasty, yet severely underrated systemic disease, which affects virtually any tissue in the body, including the brain, where it may play remarkable tricks with us.

Dodgson found this out the hard way near the end of his life. Having stayed indoors with a ‘slight hoarseness’, he had soon been laid up with what he thought to be a ‘feverish cold of the bronchial type’, an ailment he had vanquished so often that he had undoubtedly expected to do so again this time around [24]. And then, all of a sudden, his full and busy life came to a halt, leaving him dead within 10 days—probably due to pneumonia, colloquially known as ‘the old man’s friend’.⁷ At the age of 65 (almost 66), Dodgson had considered himself an old man for quite some time already, recording over and over again his sense of mortality, and his need to prepare himself to meet his Maker. And yet, those last days must have slipped by in the feverish dreams he had, leaving him only half aware of the direction his life had taken, as indicated by the disjointed final scribbles in the little notebook that was found at his bedside, which ran,

The things that I thought were a fever dream were some of them bits of 9 rule other bits of Memoria Technica & others chatty telegrams written in Memoria Technica didjits. [26]

As far as we know, these were the disorganised last words written by the man who gave the world one of the greatest literary treasures of all time—consumed by fever, and probably already on his way to meet his Maker.

7.3 All Things Considered

All things considered, what do you think should be our final verdict on the origin of Dodgson’s remarkable knowledge of perceptual distortion s? We are now in a position to answer that question—although I will be the first to admit that it is still no easy task. Throughout his life, Dodgson may well have been his own best chaperone, as suggested by one of his biographers—he certainly was his own best censor. Due to his fussiness with the diary, it is hard to tell whether the medical history reconstructed here is in any way complete. Therefore, our overall assessment of how he was affected by his various ailments and, particularly, whether any of them may have prompted him to describe the perceptual phenomena in the *Alice* book, is hampered by the realisation that the picture, as drawn by me, is almost certainly incomplete.

That said, I think that we can nevertheless conclude that Dodgson must have been familiar with the perceptual distortions he described, or else he would not have been able to come up with 13 different examples of them. Moreover, it would seem unlikely that he had based these descriptions on hearsay. Although there is no direct evidence that he had experienced them himself, throughout his life he had suffered from various conditions that are now acknowledged as factors that may trigger Alice in Wonderland syndrome. If we would draw up a list of those conditions and arrange it in accordance with their likelihood (known as a ‘differential diagnosis’ in my discipline), I think the top position would be occupied by the many infectious diseases that he had suffered from, implying that the opportunity to experience perceptual distortions—the prerequisite for describing them in the *Alice* book—may have been a blessing in disguise, an unforeseen gift of the numerous feverish episodes that he had suffered from so badly.

In second place comes migraine—to which I must add that it is a distant second place, since we do not know for sure whether Dodgson ever suffered a single attack of migraine before his 30th birthday. The third place is for intoxications of whatever kind, with alcohol being the most likely substance, as well as the one most readily available—although it could in fact have been anything, ranging from laudanum to quinine to magic mushrooms. We really do not know. Trailing far behind, in the fourth place of our differential diagnosis, is epilepsy. Whereas we know for certain that Dodgson suffered from numerous infectious diseases, experienced migraine on at least five occasions, and did indeed drink alcohol, we really have no clue as to whether he experienced a single episode of epilepsy, even though he himself had been convinced of it. As indicated, it is also the most difficult diagnosis to establish with hindsight; but, even apart from that, the descriptions of his ‘attacks’ hardly comply with tonic-clonic seizures as we know them today and as they had been known back then. Fifth, and finally, it is worth remembering our earlier observation that Dodgson seemed to have found a way to tap into the deeper layers of his consciousness, thus gaining access to areas of his psyche that would otherwise have remained out of his reach. Although he did not approach the ‘primary process’ as methodically as Dalí (with his platter and his spoon), he did patrol the borders of his waking mind—in bed, half asleep, but also during those long walks of his, around Oxford—to return from them with pieces of dialogue that seemed to have come ‘of themselves’, as he had put it. Whether during

such episodes he also experienced perceptual phenomena is something that the story does not tell. And yet, we may ask ourselves, if Dalí found imagery there, why couldn't Dodgson have done the same?

Summing up, my differential diagnosis runs as follows:

1. Infectious diseases
2. Migraine
3. Intoxications
4. Epilepsy
5. Tapping into the primary process

Obviously, this list is not set in stone. Perhaps you will be of the opinion that the order should be different—that alcohol deserves a place higher up, for example—or that items should be added or taken away. Regardless, you will by now be able to draw your own conclusions and see for yourself what you would think to be the most likely explanation.

And then what? Does that mean that we are done? Obviously, we are not done at all. Scientific research on Alice in Wonderland syndrome is only just beginning, and we have a long way ahead of us before we can say that we really understand what it is all about, and how people suffering from it should be treated. Throughout the years, I have been able to successfully treat several individuals with severe cases of Alice in Wonderland syndrome, some of whom—like Mrs. van Nuys and Ms. Rembrandt—had been plagued by it for as long as they could remember. How I would have liked to be able to boast similarly about Dimitri and Mr. Salvatore, to mention just two of the people whom I have hardly been able to help. As we have seen, there is certainly much work to be done here: In population-based research, in family studies, in genetics, in neuroimaging, in diagnostics, in the development of therapeutic protocols, and in many other areas of research. Although the road may be long, I think it is absolutely worth travelling—to expand our knowledge of the fascinating ways in which the brain deals with perceptual information and, most importantly, to be able to do something for people such as Dimitri and Mr.

Salvatore. I know that there are more people like them out there and that, once we have a better means of finding them and diagnosing them, the quality of our healthcare systems will already have improved.

References

1. Goodacre SH (1972) The illnesses of Lewis Carroll. *Practitioner* 209:230–239
2. Podoll K, Robinson D (1999) Lewis Carroll's migraine experiences. *Lancet* 353:1366 [[Crossref](#)]
3. Podoll K, Robinson D (2008) *Migraine art. The migraine experience from within*. North Atlantic Books, Berkeley
4. Hart Y (2010) Report of Dr Yvonne Hart on Carroll's neurological symptoms, August 2008. In: Woolf J *The mystery of Lewis Carroll. Discovering the whimsical, thoughtful, and sometimes lonely man who created Alice in Wonderland*. St. Martin's Press, New York, pp 298–299
5. Dodgson CL (2004) Diary entry of August 30, 1883. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson, vol 8, July 1883 to June 1892*. The Lewis Carroll Society, Luton
6. Hughes JR (2005) Did all those famous people really have epilepsy? *Epilepsy Behav* 6:115–139 [[Crossref](#)]
7. Lennon FB (1945) *The life of Lewis Carroll. Victoria through the looking glass*. Simon & Schuster, New York
8. Dodgson CL (2004) Diary entry of September 2, 1889. In: Wakeling E (ed) *Lewis Carroll's diaries. The private journals of Charles Lutwidge Dodgson, vol 8, July 1883 to June 1892*. The Lewis Carroll Society, Luton
9. Carmichael C (1996) *Wonderland revisited*. *London Miscellany* 28:19–28
10. Wakeling E (2015) *Lewis Carroll. The man and his circle*. I.B. Tauris, London
11. Reed L (1932) *The life of Lewis Carroll*. W. & G. Foyle, London
12. Hudson D (1954) *Lewis Carroll. An illustrated biography*. Constable, London
13. Leach K (2009) *In the shadow of the dreamchild. The myth and reality of Lewis Carroll*, 2nd edn. Peter Owen, London
14. Woolf J (2010) *The mystery of Lewis Carroll. Discovering the whimsical, thoughtful, and sometimes lonely man who created Alice in Wonderland*. St. Martin's Press, New York
15. Freeman R, Hambling MH (1980) Serological studies on 40 cases of mumps virus infection. *J Clin Pathol* 33:28–32 [[Crossref](#)]

16. Dejuqc N, Jégou B (2001) Viruses in the mammalian male genital tract and their effects on the reproductive system. *Microbiol Mol Biol Rev* 65:208–231
[Crossref]
17. Fitzgerald M (2004) *Autism and creativity: is there a link between autism in men and exceptional ability?* Routledge, London
[Crossref]
18. Strunz S, Schermuck C, Ballerstein S, Ahlers CJ, Dziobek I, Roepke S (2017) Romantic relationships and relationship satisfaction among adults with Asperger syndrome and high-functioning autism. *J Clin Psychol* 73:113–125
[Crossref]
19. Blom JD (2003) *Deconstructing schizophrenia: an analysis of the epistemic and nonepistemic values that govern the biomedical schizophrenia concept.* Boom, Amsterdam
20. Schmidt D, Shorvon S (2016) *The end of epilepsy? A history of the modern era of epilepsy 1860–2010.* Oxford University Press, Oxford
21. Dodgson CL (1979) Letter to Edith Blakemore, February 1, 1891. In: Cohen MN *The letters of Lewis Carroll.* Edited by Morton N. Cohen with the assistance of Roger Lancelyn Green, vol 2, ca. 1886–1898. Oxford University Press, New York
22. Centers for Disease Control and Prevention (2017) Seasonal flu death estimate increases worldwide. <https://www.cdc.gov/media/releases/2017/p1213-flu-death-estimate.html>. Accessed 22 Apr 2018
23. World Health Organisation (2017) *World malaria report 2017.* World Health Organisation, Geneva
[Crossref]
24. Dodgson Collingwood S (1899) *The life and letters of Lewis Carroll.* T. Fisher Unwin, London
25. Day D (2015) *Alice’s adventures in Wonderland decoded.* Doubleday Canada, Toronto
26. Wakeling E (ed) (2005) *Lewis Carroll’s diaries. The private journals of Charles Lutwidge Dodgson, vol 9, July 1892 to December 1897.* The Lewis Carroll Society, Luton

Footnotes

¹ It remains difficult to ascertain whether Dodgson’s letters contain any direct evidence; but, with only 10% of them having been published, there is still a chance that—someday—written evidence may surface that he did experience perceptual distortion s. However, based solely on what has been published, we must conclude that here, too, direct evidence is lacking.

² Incidentally, the way these things tend to go with people who overrate social standing, Dodgson himself had a habit of looking down on people of lower social ranks.

3 I am speculating here because it is hard to know how anyone with a fetish will enjoy their trophies, even though these are common examples of how this is done.

4 It has been estimated that Dodgson earned some £50,000 with his books and the spinoff they generated, which, corrected for inflation, would have been some £6,000,000 in 2018 [13]. Scattered over 30 odd years, that was hardly enough to make him rich, although it should have allowed him to live a bit more lavishly than he did. But that was not what Dodgson was interested in. He did not care for abundance, and he did not care for money for its own sake, either, using it instead to finance personal projects (i.e. photography, illustrations for his children's books, publishing costs, the theatre), help family, friends and acquaintances and make donations to charity organisations. He even took it upon himself to pay off the debts of an acquaintance, and invest in steamships to help out one of his cousins, losing huge sums of money in the process. What no one knew, but was discovered by Woolf when she laid hands on Dodgson's bank account of over a century old, he had made numerous overdrafts throughout his life and, by the end of his life, had spent everything that he had had, leaving behind an account that was once more overdrawn [14].

5 In fact, in my line of work, I see a relatively large number of men who have developed breasts, mostly as a side effect of the antipsychotic medications they are using, which may increase the production of prolactin, the pituitary hormone responsible for the development of female breast tissue.

6 'Aura' is the name for a symptom preceding or accompanying a seizure, usually of a perceptual nature, such as a hallucinated smell or taste, an aberrant bodily sensation, a vision or—sporadically—hallucinated music or other sounds. Such phenomena are far from obligatory in epilepsy, but had they been present, they would have made a seizure a bit more likely.

7 It has traditionally been assumed that Dodgson did indeed die of pneumonia, as suggested by his physician, Dr. Dabbs; however, here, too, we should keep an open mind and take other possibilities into consideration. For example, Dodgson was one of the first to have an asbestos gas fire installed in his house because it was considered more cost-efficient and 'cleaner' than traditional coal fires and could, moreover, be left burning day and night without need of attendance [25]. Since the carcinogenic effects of asbestos had not even been recognised in Dodgson's time, Dabbs cannot be expected to have taken them into account while establishing the cause of death.

Appendix A

Table A.1 Charles Dodgson 's medical history

• Stammering (lifelong)
• Prosopagnosia (possibly lifelong)
• Childhood: infantile fever , complicated by hearing impairment in the right ear
• 1849: pertussis (whooping cough)
• 1850: mumps , complicated by permanent deafness in the right ear; possibly also complicated by orchitis ?
• 1855: influenza
• 1855: forehead cut open due to a fall while attempting to skate
• 1856: visit to the oculist, Dr. William Bowman , about an unspecified problem with the right eye
• 1856: called on the aurist, Dr. Joseph Toynbee , because of continuing deafness in the right ear
• 1856: swollen face after ascent of Great Gable in icy cold wind; possibly ‘ neuralgia’
• 1857: stayed in, ‘not feeling well’
• 1860: sought the assistance of Dr. James Hunt because of stammering; tried vocal exercises
• 1862: had a tooth stopped by the dentist, Mr. Bevers
• 1863: had himself vaccinated against smallpox
• 1866: ‘sort of rheumatic attack in the neck and shoulders’, keeping him in for several days
• 1866: ‘neuralgia’ shifted from the neck to the face, causing several days of considerable pain; ascribed by Dodgson to the cold weather and a hollow tooth; had the tooth stopped, and weather became warmer, after which the pain stopped. Believed it to have been ‘ neuralgia suborbitalis ’
• 1867: ‘taken ill’
• 1867: procured salve <i>fortic-doloreux</i> ; unsure whether for himself
• 1870–1871: bad cough (2 weeks)
• 1871: had himself vaccinated against smallpox for the second time
• 1871: sore throat and a headache, followed by chilliness in the evening and cough at night; stayed in for 12 days
• 1872: attended a lecture by Dr. Lewin on stammering , and subsequently tried out his ‘system’ by reading out loud, together with a fellow-stammerer, Mr. Hine
• 1873: interview in London with Mr. Rivers , seeking a cure for his stammer
• 1874: interview in London with Mr. Rivers, together with his sisters, Elizabeth and Caroline, seeking a cure for their stammer
• 1874: stayed in, suffering from ‘ neuralgia’

• 1874: a cold on the throat and chest
• 1874: stayed in, 'not feeling well'
• 1876: gathering on the right heel
• 1877: called on the homoeopath, Dr. Edward Shuldham , for unknown reasons
• 1878: called on the homoeopath, Dr. Edward Shuldham, for unknown reasons
• 1878: a cold on the throat and chest
• 1878: treated a cold that had kept him in for several days with homoeopathic doses of aconite and arsenic for 2 days; mentions 'a bad cold all week'
• 1880: treated a cold with homoeopathic doses of aconite, mercury solution, and white oxide of metallic arsenic
• 1881: voice nearly reduced to a whisper; recovered within a week
• 1881: kept to his rooms for a week after a 23-mile walk on a hot day, which gave him 'a sort of mixture of sun stroke and ague '; believed that it was 'an attack of " vesical catarrh " with fever as a secondary'
• 1881: feverish symptoms, possibly due to "vesical catarrh" (i.e. cystitis), treated with quinine by Mr. E.L. Hussey
• 1881: eight visits to the dentist, Mr. Whatford, who did 'a quantity of stopping'
• 1882: seasick on the <i>Solway</i> , en route to Greenock
• 1882: visit to the dentist, Mr. Whatford, to have a tooth stopped. Had to go back for two more stoppings
• 1882: oval patch under one arm: thinks it is some form of erythema , although the homoeopath, Dr. Shuldham , called it tinea . Shuldham prescribed sulphurous acid , but Dodgson rather took ' Graphites (internally) and Calendula (externally)'
• 1883: stayed in 4 days with a severe cold in the head
• 1883: fever and 'a sort of ague ', possibly due to cystitis , followed by a relapse, keeping him in for more than 3 weeks. Says that he is 'ill with bilious fever '
• 1883: fever ('much like my last attack') and a headache , stayed in for 5 days
• 1884: 'an ague-like cold fit'; lungs examined by Dr. James Beddard , who pronounced him a 'thoroughly healthy man', diagnosing 'a feverish cold of ague-type'
• 1885: experienced 'for the second time that odd optical affection of seeing moving fortifications , followed by a headache'. N.B. This is the first mention of such an attack in Dodgson's diaries
• 1886: an 'attack', followed by 'a sort of headache'; does not feel his usual self for a week or 10 days. Thinks it might have been epilepsy
• 1886: 'not well'
• 1886: one of his 'ague-like feverish attacks', 5 days in bed, total duration 2 weeks; sent for the surgeon, Dr. Arthur Sherwood
• 1886: ' lumbago , or sciatica, or perhaps a mixture of the two'
• 1887: 'a bad cold'

• 1887: ‘fever of ague-type’, for at least 5 days
• 1888: synovitis of the right knee, as diagnosed by Dr. Doyne , who bandaged the knee from the foot upwards after painting it with iodine; stayed in bed and on the sofa for more than 6 weeks; got an elastic knee-cap after 1 month
• 1888: on getting up, saw fortifications in the right eye only, at the outer edge, without a headache
• 1888: went to Dr. Paget in London to get advice about his ailing knee (almost 3 months after the start of the ‘ synovitis’)
• 1888: consulted the homoeopath, Dr. Burnett , who thought that Dodgson’s spleen was out of order and prescribed “<i>Rubia tinctorum</i> ”, as the probable cause of eczema and varicosis ’
• 1888: optical fortifications, beginning ‘with a distinct loss of a large piece of the area of vision of the left eye, the “blind” patch being in the right-hand corner, just where, directly afterwards, the fortifications appeared’
• ±1889: bitten by a dog
• 1889: a boil on the left wrist, scarcely healed for a month
• 1889: ‘synovitis’ in the left knee, treated with bandaging and iodine; laid up for a week
• 1889: fortification without a headache
• 1889: operated on by Dr. Sherwood , removing a small fleshy excrescence (probably on the left wrist) after locally applying cocaine first
• 1890: ‘laid up for a week with “synovitis” in left knee (probably rheumatic)’
• 1890: 2 weeks indoors because of ague , cystitis , lumbago and headache
• 1890: laid up on the sofa because of ‘synovitis’ of one knee; painted it with iodine and bandaged it
• 1891: loss of consciousness for a duration of 1 hour, followed by a headache. Thinks it was an epileptic attack; kept feeling unwell for more than 4 months and considered himself seriously ill; headache lasted for more than 10 months
• 1891: saw fortifications; possibly had a déjà- experience 1 week beforehand
• 1891: lumbago and ‘synovitis’ in the left knee; bandaged the knee and painted it with iodine; lived ‘like a hermit’; tried ‘massage’ and a little walking; in all, duration of more than 3 months
• 1891–1892: laid up on the sofa for more than 2 weeks with a bad knee (‘of the nature of “housemaid’s knee”’), keeping it up in a horizontal position, painting it with iodine
• 1892: diarrhoea for 4 or 5 days, put himself under Dr. Sherwood ’s directions
• 1892: small gumboil opposite to a double tooth, and pain in the front teeth (possibly due to a cold or flu ?)
• 1892: robbed of voice for 10 days, stayed in with a bronchial cold
• 1893: stayed in because of lumbago
• 1893: after a week indoors, took Dr. Brook ’s medicine ‘to set the liver, etc., right’
• 1894: ague -like cold, keeping him in for more than 10 days, took quinine again

• 1894: pain in the right shoulder, supposed to be ‘rheumatic’ in nature, and pain behind false ribs on that side, ‘suggestive of pleurisy ’; 1 day later a familiar pattern of ‘ague’ in its ‘three usual stages’; duration of 2 or 3 days
• 1895: stayed in for 3 weeks due to ‘a bad cold’, ‘almost bronchitis ’, ‘ influenza ’, with a temperature rising to 39 °C; had feverish dreams; lingered on for 10 more days
• 1897: lost right front tooth
• 1898: feverish cold of the bronchial type; died within 10 days, possibly of pneumonia . NB: had owned an asbestos gas fire for the last 10 years of his life, which he kept on burning day and night during the winter

Table A.2 Visual distortions (metamorphopsias) that may occur in the context of Alice in Wonderland syndrome (after Blom 2017)

Type of metamorphopsia	Characterisation	Key reference
Achromatopsia	The inability or strongly diminished ability to perceive colour	Zeki (1990)
Akinetopsia	The inability to perceive motion	Zeki (1991)
Arugopsia	Seeing wrinkled or rough surfaces as smooth	Blom (2017)
Chloropsia	Green vision	Pinckers et al. (1989)
Chromatopsia	Seeing things in a single hue (as in chloropsia, cyanopsia, erythropsia, ianothinopsia and xanthopsia)	Pinckers et al. (1989)
Complicated metamorphopsia	A metamorphopsia which alters the affective assessment of the extracorporeal environment, rendering it either beautiful, ugly or frightening	Critchley (1953)
Corona phenomenon	An extra contour around objects	Klee and Willanger (1966)
Cyanopsia	Blue vision	Pinckers et al. (1989)
Dyschromatopsia	Colour confusion	Zeki (1990)
Dysmegalopsia	A diminished ability to appreciate the size of objects	Wilson (1916)
Dysmetropsia	A change in the apparent size and distance of objects	Wilson (1916)
Dysmorphopsia	Lines and contours appearing to be wavy	Lunn (1948)
Dysplatopsia	Objects appearing flattened and elongated	Wieser (2000)

Type of metamorphopsia	Characterisation	Key reference
Enhanced stereoscopic vision	An exaggeration of the depth and detail of visually perceived objects	Critchley (1949)
Entomopia	Seeing multiple images, as if perceived through an insect's eye	Lopez et al. (1993)
Erythropsia	Red vision	Pinckers et al. (1989)
Gyropsia	Seeing an illusory, circular movement	Ey (1973)
Hemimetamorphopsia	A visual distortion of only one half of an object	Nijboer et al. (2008)
Hyperchromatopsia	Colours seen as exceptionally bright	ffytche and Howard (1999)
Ianothinopsia	Purple vision	Pinckers et al. (1989)
Illusory splitting	A vertical splitting of objects	Podoll and Robinson (2002)
Illusory visual spread	A perceived extension, expansion or prolongation of objects	Critchley (1949)
Inverted vision	Objects appearing rotated (usually in the coronal plane, over 90° or 180°)	Winslow (1868)
Kinetopsia	Illusory movement	Ey (1973)
Loss of stereoscopic vision	Objects appearing two-dimensional or 'flat'	Critchley (1949)
Macroproxioptia	Objects appearing larger and closer by than they are	Critchley (1953)
Macropsia	Seeing things larger than they are	Critchley (1949)
Micropsia	Seeing things smaller than they are	Critchley (1949)
Microtelepsia	Objects appearing smaller and further away than they are	Taylor et al. (2003)
Monocular metamorphopsia	Metamorphopsia for one eye	Willanger and Klee (1966)
Mosaic vision	A fragmentation of perceived objects into irregular, crystalline, polygonal facets, interlaced as in a mosaic	Sacks (1970)

Type of metamorphopsia	Characterisation	Key reference
Palinopsia	Illusory reoccurrence of visual percepts (as in polyopia, illusory visual spread and the trailing phenomenon)	Critchley (1949)
Pelopsia	Objects appearing closer by than they are	Ey (1973)
Plagiopsia	Objects appearing as if tilted	Critchley and Ferguson (1933)
Polyopia	Seeing multiple identical copies of a single image	Klüver (1966)
Porropsia	Stationary objects appearing to move away	Vujić and Ristić (1939)
Prosopometamorphopsia	Apparent distortion of faces	Bodamer (1947)
Simple metamorphopsia	A metamorphopsia which does not alter the affective assessment of the extracorporeal environment	Critchley (1953)
Teleopsia	Objects appearing to be further away than they are	Wilson (1916)
Trailing phenomenon	A series of discontinuous stationary images trailing behind a moving object	Asher (1971)
Visual allachaesthesia	Objects appearing dislocated into the opposite visual field	Beyer (1885)
Visual perseveration	An illusory reoccurrence of visual percepts after an object has moved out of focus	Critchley (1949)
Xanthopsia	Yellow vision	Pinckers et al. (1989)
Zoom vision	Vision fluctuating between micropsia and macropsia, or between microtelepsia and macroproxiopia	Sacks (1970)

Table A.3 Somaesthetic and other non-visual distortions that may occur in the context of Alice in Wonderland syndrome (after Blom 2017)

Type of distortion	Characterisation	Key reference
Aschematia	Inadequate representation of the space occupied by some part of the body	Bonnier (1905)
Derealisation	Experiencing the world as unreal	Mayer-Gross (1935)
Depersonalisation	Experiencing oneself as unreal	Dugas (1898)

Type of distortion	Characterisation	Key reference
Hyperschematia	Overrepresentation of the space occupied by some part of the body	Bonnier (1905)
Hyposchematia	Underrepresentation of the space occupied by some part of the body	Bonnier (1905)
Illusory feeling of levitation	Sensation of floating in the air	Todd (1955)
Palisomesthesia	Illusory reoccurrence of somesthetic percepts	Feldman and Bender (1970)
Paraschematia	Inappropriate representation of the space occupied by some part of the body	Bonnier (1905)
Partial macrosomatognosia	Experiencing a part of the body as larger	Frederiks (1963)
Partial microsomatognosia	Experiencing a part of the body as smaller	Frederiks (1963)
Protracted duration	Deceleration of psychological time	Hoff and Pötzl (1934)
Quick-motion phenomenon	Acceleration of psychological time	Hoff and Pötzl (1934)
Splitting of the body image	Sensation of one's own body being split in two, usually down the middle	Podoll and Robinson (2002)
Time distortion	Altered experience of psychological time	Hoff and Pötzl (1934)
Total body macrosomatognosia	Experiencing the whole body as larger	Frederiks (1963)
Total body microsomatognosia	Experiencing the whole body as smaller	Frederiks (1963)

Table A.4 Conditions in the context of which symptoms of AIWS have been described (after Blom 2017)

Conditions	Key references
<i>Infectious diseases</i>	
Acute disseminated encephalomyelitis	Coven et al. (2013)
Coxsackie B1 virus encephalitis	Wang et al. (1996)
Cytomegalovirus	Losada-Del Pozo et al. (2011)
Epstein-Barr virus encephalitis (infectious mononucleosis)	Piessens et al. (2011)
H1N1 influenza virus encephalitis	Augarten and Aderka (2011)
Influenza A virus encephalitis	Kuo et al. (2012)

Conditions	Key references
Lyme neuroborreliosis	Binalsheikh et al. (2012)
Malaria	Kadia et al. (2017)
Meningococcal meningitis	Trolle (1951)
Neurosyphilis	Wilson (1916)
Scarlet fever	Brumm et al. (2010)
Typhoid encephalopathy	Kitchener (2004)
Varicella zoster encephalitis	Soriani et al. (1998)
<i>Neurodegenerative disorders</i>	
Creutzfeldt-Jakob disease	Naarden et al. (2019)
<i>Central nervous system lesions</i>	
Brain tumour	Geyer (1963)
Cavernous angioma	Philip et al. (2015)
Cerebral arteriosclerosis	Veraguth (1903)
Cerebral thrombosis	Critchley (1949)
Creutzfeldt-Jakob disease	Naarden et al. (2019)
Haemorrhagic stroke	Camacho Velasquez et al. (2015)
Ischemic stroke	Camacho Velasquez et al. (2015)
Microembolisation following open heart surgery	Meyendorf (1982)
Robin Hood syndrome	Morland et al. (2013)
Traumatic encephalopathy	Willanger and Klee (1966)
Wallenberg's syndrome	Bjerver and Silfverskiöld (1968)
<i>Peripheral nervous system lesions</i>	
Eye disease	Bay (1953)
Labyrinthine disease	Deecke et al. (1981)
<i>Paroxysmal neurological disorders</i>	
Epilepsy	Heo et al. (2004)
Headache with neurological deficits and CSF lymphocytosis	Zeiner et al. (2015)
Migraine	Ilik and Ilik (2014)
<i>Psychiatric disorders</i>	
Delusional misidentification syndrome	Takaoka et al. (2001)
Depressive disorder	Bui et al. (2010)
Derealisation/depersonalisation disorder	Wiese (1979)

Conditions	Key references
Dissociative disorder	Lipsanen et al. (1999)
Schizoaffective disorder	Blom et al. (2011)
Schizophrenia	Coleman (1933)
<i>Medications</i>	
5-HT ₂ agonists	Dubois and Vanrullen (2011)
Cough medicine (containing dihydrocodeine and <i>dl</i> -methylephedrine)	Takaoka and Takata (1999)
Dextromethorphan	Losada-Del Pozo et al. (2011)
Montelukast	Bernal Vañó and López Andrés (2013)
Oseltamivir	Jefferson et al. (2009)
Topiramate	Evans (2006)
<i>Illicit substances</i>	
Amanita muscaria	Brvar et al. (2006)
Amphetamines	Curran et al. (2004)
Ayahuasca	Critchley (1929)
Cannabis	Losada-Del Pozo et al. (2011)
Cocaine	Unnithan and Cutting (1992)
LSD	Lerner and Lev Ran (2015)
MDMA	Litjens et al. (2014)
Mescaline	Klüver (1966)
Toluene-based solvent	Takaoka et al. (2001)
Trichlorethylene	Todd (1954)
<i>Miscellaneous</i>	
Hyperpyrexia	Eshel et al. (1987)
Hypnagogic state	Jürgens et al. (2011)
Hypnopompic state	Schneck (1971)
Hypnotherapy	Schneck (1969)
Sensory deprivation	Heron (1961)

Appendix B

***On Catching Cold* , by Charles Dodgson (University Press, Oxford, 1881)**

ON CATCHING COLD.

[Extracted from Dr. Inman's books, 'The Restoration of Health', and 'The Preservation of Health'.]

'The most common cause of catarrh is a sudden transition from a moist and cold atmosphere—such as is commonly met with in an "open" English winter—to a hot and dry room; and those people are most subject to "bad colds" who by accident or design have to undergo such transitions. For example, a lady fresh from a ball-room drives home a good long distance on a nasty night in winter. In spite of a comfortable carriage, she respires the cold air of December or January, and arrives home jaded with dancing and chilled by the night dews. Joyfully she rushes to her comfortable boudoir to find warmth, quiet, and a pleasant nook for chat. But she soon finds that she has "caught a cold"—it may be a fatal one—and then she and her friends lay the blame at the door of the chill on leaving the assembly-room, rather than to the comfort of the chamber of luxury. From long personal experience I would say that no one single cause is more frequently in operation to produce catarrh than the one referred to, and I entirely agree with the remark of an old surgeon, that it would be more sensible for individuals to say they had been "catching hot" when they felt themselves "in" for a catarrh, than to say that they had caught cold'.

The Restoration of Health, p. 75.

'On this point we have personal experience. During many successive winters the present writer had to turn out at about five o'clock in the evening, and lecture upon "medicine" until six. His day's work then being really over, he retired to his "study," which was as warm and comfortable as a doctor could desire. Yet, over and over again, the return home was attended by the uncomfortable sensation of "having caught a cold". In vain the mind was cudgelled to determine how the catarrh had been produced. The bare fact remained, and as the winter advanced the attacks became so severe and constant that it became a question how far they were dependent upon some serious constitutional change. At length, whilst preparing a lecture upon "common colds," the author found it stated by Dr. Copland

that their most frequent cause was coming into a hot and dry atmosphere after being exposed to a moist cold air. Now this was the very thing that “poor Pilgarlick” had habitually done. He had flown to his warm sitting-room as soon as the front door enabled him to escape the moist cold air of an English winter, and, as a result, he had taken “hot” instead of catching “cold.” He changed his plan forthwith, and gradually accustomed himself to the chillier rooms of his house before he went into the warm one, and since then he has escaped the severe catarrhs, coryzas, or influenzas which once tormented him. A fact like this led him to philosophise. He thought of chilblains, and recollected that the most common cause of them is a rapid transition from the cold of ice and snow to the warmth of a fire and hot water. The schoolboy cares little for itching toes so long as he is skating or snowballing, but he suffers from them as soon as he has placed his feet on the fender before the fire. It is clear, then, that the cold has injured the parts by weakening the blood-vessels and depriving the solid tissues of their blood; but, so long as the cold continues and the flesh is not “frost-bitten,” the injury is not recognised. When, on the other hand, the heat is restored, it is at once apparent that the blood-vessels, having had their natural vitality impaired, are abnormally dilated, and the circulation of blood arrested, not because none is there, but because its channels are unusually distended, resembling lakes rather than rivers. In medical language, the parts are “congested.” So it is in catarrh: the chilled nose, throat, and chest are starved by a moist and cold air whenever a person is for a long time exposed to its influence; and, if the individual suddenly comes to a hot and dry atmosphere, a similar change occurs to that which takes place in the hands and feet. Thus, sore-throat and catarrh may frequently be considered as a sort of chilblain of the fauces and the air-passages’.

The Preservation of Health, p. 161.

[The following passage, referred to in p. 2, occurs in Dr. Copland’s ‘Dictionary of Practical Medicine’, Article ‘Catarrh’.]

‘Sudden change from a low to a high temperature... will often produce catarrh. This is especially the case, if the exposure to warmth be sudden, after an impression of cold of some continuance, as the coming into an over-heated apartment out of a cold and moist atmosphere,—the

instantaneous transition from a raw air of about 32° to a dry air of upwards of 70°’.

Appendix C

Proposed Diagnostic Criteria for Alice in Wonderland Syndrome

Alice in Wonderland Syndrome

1. Alice in Wonderland syndrome, central type , in the context of another medical condition (to be specified if known);
2. Alice in Wonderland syndrome, peripheral type , in the context of another medical condition (to be specified if known); or
3. Alice in Wonderland syndrome not otherwise specified (NOS)

Diagnostic Criteria

(A). The presence of one or more of the following perceptual distortions, currently or in the past. Note that other types of distortion, not yet described in the literature, may also count.
Achromatopsia
Akinetopsia
Arugopsia
Aschematia
Chloropsia
Chromatopsia
Corona phenomenon
Cyanopsia
Dyschromatopsia
Dysmegalopsia
Dysmetropsia
Dysmorphopsia
Dysplatopsia
Enhanced stereoscopic vision
Entomopia

Erythropsia
Gyropsia
Hyperchromatopsia
Hyperschematia
Hyposchematia
Ianothinopsia
Illusory feeling of levitation
Illusory splitting
Illusory visual spread
Inverted vision
Kinetopsia
Loss of stereoscopic vision
Macroproxiopia
Macropsia
Micropsia
Microtelepsia
Mosaic vision
Palinopsia
Palisomesthesia
Paraschematia
Partial macrosomatognosia
Partial microsomatognosia
Pelopsia
Plagiopsia
Polyopia
Porropsia
Prosopometamorphopsia
Protracted duration
Quick-motion phenomenon
Splitting of the body image
Teleopsia
Total body macrosomatognosia
Total body microsomatognosia

Trailing phenomenon
Visual allachaesthesia
Visual perseveration
Xanthopsia
Zoom vision
(B). For a significant portion of time since the onset of symptoms, a marked disturbance of functioning in one or more major areas of living, such as work, education, play, interpersonal relations or self-care
(C). Optional: the presence of adjuvant symptoms, i.e. derealisation or depersonalisation
<i>Specifiers:</i>
• <i>Specify complexity</i>
– <i>Simple (unimodal)</i>
– <i>Complex (multimodal)</i>
• <i>Specify duration since onset</i>
• <i>Specify course:</i>
– First episode, intermittent
– First episode, continuous
– First episode, currently in remission
– Multiple episodes, intermittent
– Multiple episodes, currently in remission
• <i>Specify central or peripheral type:</i>
– Central type
– Peripheral type
• <i>Specify underlying medical condition:</i>
– Present (specify name of disorder)
– Absent (score as Alice in Wonderland syndrome not otherwise specified)

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